THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—13TH YEAR. SYDNEY: SATURDAY, SEPTEMBER 18, 1926. No. 12.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.

"The Leprosy Problem," by E. H. MOLESWORTH, M.B. 365

"Tuberculoid Leprosy," by A. H. TEBBUTT, D.S.O., V.D., B.A., M.B., D.P.H. 381

REPORTS OF CASES—

"Hydatid of the Lung," by E. H. STOKES, M.B., Ch.M., with a Note on the Serological Findings, by C. H. SHEARMAN, M.B., Ch.M. 386

REVIEWS—

Goitre 388

LEADING ARTICLES—

Segregation for Infective Diseases 389

CURRENT COMMENT—

Sympathetic Innervation and Skeletal Muscle Tonus 390

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.

Morphology 392

Morbid Anatomy 392

BRITISH MEDICAL ASSOCIATION NEWS—

Medico-Political 394

Scientific 399

Nominations and Elections 400

MEDICAL APPOINTMENTS VACANT, ETC. 400

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 400

DIARY FOR THE MONTH 400

EDITORIAL NOTICES 400

THE LEPROSY PROBLEM.[1]

By E. H. MOLESWORTH, M.B. (Sydney),

Honorary Physician for Diseases of the Skin, Royal Prince Alfred Hospital, Camperdown, Sydney; Lecturer in Diseases of the Skin, The University of Sydney.

Introduction.

I WISH first of all to express my appreciation of the opportunity afforded me of addressing our Victorian colleagues. And I am grateful not only for the opportunity of speaking to the Association, but of getting directly within hearing of the Commonwealth Government and its medical representatives.

And although I fear that you may think that my subject, "Leprosy" is an unpromising and uninteresting one, I am vain enough to hope that I may be able to inspire you to some degree with my interest in the subject and I trust that the efforts of Dr. Tebbutt and myself tonight will arouse fresh activity, directed toward the better management of the problem of leprosy and more humanitarian treatment of the unfortunate sufferer from the disease.

I must confess that Dr. Tebbutt unlike myself is a very retiring man, more likely to shut himself up in his laboratory than to invade the medical stronghold of another State and that I have bullied him into buckling on his armour and taking the field. However, once on the warpath, he is a doughty fighter as I very well know, we being very old and friendly enemies. In fact I invade his lair in the Pathology Department at the Royal Prince Alfred Hospital on frequent occasions and we there wage the eternal warfare of the clinician and the pathologist. I accuse the Pathological Department of thinking in their heart of hearts: "Oh, another specimen from Molesworth—he is always looking for leprosy or actinomycosis or some such weird thing—bother his enthusiasm." This Tebbutt indignantly denies. However, just at present I am in the position of victor and while in that happy position I am able to drag from his abysmal haunts the hermit of our hospital and make him show you some of his excellent work.

As I have said, we are very friendly antagonists and he will, I am sure, not object to the manner of introduction, especially as he knows I am very appreciative of all the help he has given in this matter.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 7, 1926.

I also wish to thank Dr. Dew about whom I shall speak later and Dr. Cumpston who, when I wrote to him telling him of my intention to read this paper, immediately encouraged me and gave me all the references and information at his immediate disposal. Also Dr. Taylor of whom more anon has helped me considerably in the preparation and illustration of my paper and I wish to thank him. I want to thank Dr. Moran for his ever present help in hunting through his extensive library for references to the subject of leprosy and for lending me a number of books dealing with the subject.

One thing more and I shall be ready to begin. Drs. Stephens and Devine have come over to us in Sydney and have acted as a spur to our idealism and activities. Devine said the last time I spoke to him in Sydney: "Some of us are not content to cease to be students when we graduate."

This is the spirit to breathe into the nostrils of our profession and I was thereby stimulated to classify and correlate a number of ideas which had been simmering for a long time in my consciousness.

As to Dr. Stephens he does not even need to speak to express energy and inspiration.

The interchange of ideas and ideals which they initiated, I thought we might continue, if I could persuade Tebbutt to come over. And I did and here he is.

With apologies for this long introduction I will now open my subject.

Infectivity and Bacterial Origin.

There are several interesting aspects of the leprosy problem, a subject which has been allowed to lapse for an unduly long time in Australia since the end of the controversy as to the infectivity and bacterial origin of the disease.

Leprosy was common in the ancient civilizations (hence the name *Elephantiasis Græcorum*) and during the Middle Ages plagued Europe. At the present time among Europeans the disease is rare except in Scandinavia and in Spain, where it is still relatively common.[1]

Why is it that diseases which were at one time prevalent in a particular race and country, die out if only to make room for others? All infective diseases do so, as we can observe today in the case of tuberculosis. I well remember a dramatic moment at the Lister Institute. Afternoon tea time was a social event utilized by Professor Martin to encourage exchange of ideas in order to prevent specialists from getting too deep in their own rut. With this object in view he used to lead the conversation into a chosen channel and guide it in a manner that enabled him to spring a surprise at the psychological moment. One afternoon we came into the room to find the walls festooned with charts, wall tables of graphs and curves and everyone knew that some such scheme was in preparation. The subject introduced was tuberculosis and after some general discussion Professor Martin asked whether the company assembled agreed that the decline in the incidence and death rate of tuberculosis was largely due to the institution of hygienic measures of control and prophylaxis and open air methods of treatment. This was agreed to by a number of those present and I am afraid I was of those foolish ones who had forgotten to keep their lamps replenished with the oil of knowledge. Then the professor sprung his mine. Having fixed the date of the first institution of such measures, he then proceeded to show conclusively by his graphs and curves that long before that time there had been a steady and regular drop in the incidence and death rates of tuberculosis and that the institution of such measures was but an artificial mark on the graph with no influence upon the curve. And so it continues—the situation is humiliating but true that tuberculosis is diminishing because it is killing out the susceptible stock.

Now it will be admitted that the diseases tuberculosis and leprosy are closely cognate, the organisms being especially nearly related. And as it is with tuberculosis, so must it be admitted to be in the case of leprosy, unless strong and numerous reasons can be put forward against this conclusion.

It is admitted that the elimination of susceptible stock is the manner in which infective diseases die out in a community and the onus of proof that a particular disease, admitted to be infective, dies out as a result of other causes lies with the authors of the new hypothesis.

In reading a new book by Rogers and Muir on leprosy I was much struck by the neglect of this accepted explanation of the decline of leprosy among European races, the credit for which was given entirely to segregation and control. In the case of the greater part of Europe at least these regulations were designed and administered in the Middle Ages. In England the leper was divorced, forced to wear a bell around his neck in the street, forbidden to handle foodstuffs and accommodated in leper houses *et cetera*. But he was at liberty to wander about the country and was forced to beg his bread.

Looking at these measures from an entirely unprejudiced standpoint it is hard to imagine that they could seriously diminish the incidence. The disease would not be recognized at such a period of history, except in its most obvious and advanced stages, when it had most probably been in existence for more years than remained to the sufferer and even during these remaining years there existed a hardly diminished chance of infection of others by the mendicant lepers who wandered about the country and who probably wore the bell and obeyed the regulations only in districts where they were known and when it suited them for the purpose of begging.

For a description of the leper houses of the Middle Ages, the manner of their management and conduct and the degree of isolation afforded by them I must refer you to Garrison's "History of Medicine," page 162, Virchow's *"Zur Geschichte des Aussatzes und der Spitälen,"* [2] the prize essays on leprosy by Newman,[3] but more especially to the work of Cabanès (*"Mœurs intimes du Passé," Cinquième Série, "Fléaux de l'Humanité"*). It is sufficient

here to say that patients were not confined in them, merely housed and to some extent supported, allowed to beg but compelled to share their spoils, while married men were allowed to visit their wives (in France), but were not allowed more than three hours of the day for this purpose.[4]

Newman does not consider that leper houses acted or were designed to act as isolation hospitals, but simply as charitable centres and quotes Sir James Y. Simpson in support. Jonathan Hutchinson[5] says it is "an utter mistake to believe that efficient segregation was practised."

Thus it must be admitted that the contention that the decline of leprosy was due to the enactments in the Middle Ages and the segregation achieved by the leper houses (which by the way accommodated only a part of the known leper population, not to speak of the undiscovered lepers) rests upon perilously slender evidence. While not denying the influence of isolation if efficiently performed, it is difficult to understand how the theory that leprosy declined in Europe as a result of segregation can be entertained at all, when the constitution and conduct of leper houses are studied. Rather would this study make it apparent that, segregation being neither the object nor the effect of these institutions, some cause other than segregation must perforce be sought to explain the decline of leprosy in Europe.

In preparing estimates of the numbers of lepers in different countries at different periods Rogers and Muir admit that the numbers are not even approximate, because of the concealment by relatives. How much more so must this have been the case in the Middle Ages? And how many cases of other diseases of the skin must have been mistaken for leprosy?

These and other factors make the deductions from such figures highly untrustworthy and practically useless.

One fact can be determined sometimes with reasonable exactitude and that is the dates of obvious contamination of the race and the practical disappearance of the disease.

In England this period is A.D. 600 to 1600, but odd corners like Cornwall and north Scotland, Shetland Isles *et cetera* were infected three or four hundred years later and in these localities the disease has only just lately died out. Taking European communities generally this rule holds: "Late come, late go." The probable explanation of the persistence of leprosy in certain countries or districts is that communities living secluded in the *fjords* of Norway or the mountains of Spain escaped infection for a long time, as did the inhabitants of Cornwall who until quite recently called all other Englishmen foreigners. But though they escaped by their isolation for a long time, they have had to run the gauntlet for as long a period as the less sheltered communities. Hence it is that leprosy has only recently disappeared or is still to some extent endemic in these countries.

The heaviest incidence in the world at present is in equatorial Africa and it has been stated that Africa has been infected since 4000 years B.C. But it must be remembered that this was the Africa of the old world civilization, cut off by the Sahara and the ocean from equatorial and south Africa. Egypt and north Africa have only a small leprosy incidence, as might be expected under this theory. But equatorial Africa according to Mungo Parke and Moore[1] was infected with leprosy only since the opening up of the country and the introduction by the Mohammedan Arabs of the Soudanese slaves. Hence it is probable that the infection of equatorial Africa is only recent historically and the conditions of the climate, nutrition, housing and uncleanness being all in favour of the disease (which in addition is favoured by the fact that the race is new to the attack) explain the exceptionally high incidence today.

The cases of China and India are not the same. Here the disease is certainly much older, but there is no real proof that it is much older than in Asia Minor and southern Europe. It has been stated that China was infected a thousand or more years B.C., but the evidence of this in Chinese writings is so vague as to be valueless.[6] Rogers and Muir say China is believed to have been infected for over two thousand years,[1] but if the disease is accepted as arising in Egypt[6] and as only reaching Rome with Pompey's legions in 62 B.C., even this is difficult to believe.

The evidence of the extreme antiquity of leprosy is very slender. For example, the prescription for leprosy in Egypt 4600 B.C.[6] is very shaky, when we consider that the leprosy of the Old Testament almost certainly included psoriasis and leucoderma if it ever really included what we know as leprosy. The Hebrew *Zaraath*[6] [7] and the *Kusta*[1] of the Veda in India in 1400 B.C. and the disease for which the Egyptian prescription in 4600 B.C. was designed, were all most likely dermatoses credited with being or in reality infectious, but not as likely to be leprosy as ringworm, psoriasis and leucoderma or chronic lichenification from eczema. This is supported by the fact that the lepra of Hippocrates (400 B.C.) is admitted to be psoriasis, though the name has been transferred to what we know as leprosy, but which was unknown to Hippocrates. Leprosy is said to have been described by Aristotle in 354 B.C.[6] This confusion, vagueness of description and transference of names make it only too fatally easy to proclaim the ultraantiquity of leprosy in any country and should make us exceedingly careful of accepting the shadowy evidence put forward.

In any case, when we come to deal with the incidence in other than European communities, the isolation of tribes, of castes, of religions have to be given deep consideration. And, in addition, where it must be admitted that individuals of the same race show great variation in resistance to infection, so I think it must be agreed that different races may and do show varying resistance and as the Spaniard may be less resistant than the Englishman, so the Asiatic may be more susceptible than the European.

Moreover, it is a well known bacteriological fact that if a series of highly susceptible animals be inoculated with a particular strain or culture of bacteria, the virulence of this strain is appreciably or even greatly increased. Conversely, if a series of resistant but not wholly immune animals be inoculated, the virulence of the organism is diminished. Now, visualizing the infection of races with Hansen's bacillus and admitting racial, as one must admit individual variations in resistance, we can easily understand the difference in the length of time required for the development of racial immunity up to the point of determining the disappearance of the disease.

Also it is understandable that a comparatively resistant European can contract the disease from an infected person of a susceptible race like an African or Polynesian or Australian aboriginal since the passage through a highly susceptible host will have raised the virulence of the organism to a pitch which may enable it to overcome the resistance of the European.

The development of racial immunity or insusceptibility is simple enough to explain—the application of the well-known law of evolution, the survival of the fittest.

When a disease attacks a community or a nation those individuals or families which have the least natural resistance, are most severely and most frequently attacked. And as it seems undoubted that childhood and adolescence are the periods of greatest susceptibility to infection with leprosy and as leprosy profoundly diminishes the reproductive capacity, the susceptible stock is killed out. If the restriction on lepers by adding hardship, starvation and exposure such as resulted from mediæval regulations, shortened the life of the leper and as Liveing suggested[1] acted by killing out the race of lepers, that I would suggest was the greatest effect to which the regulations contributed.

Notice is drawn by some[6] to the curiously rapid disappearance of leprosy in the sixteenth century and to the coincidence of its disappearance with the arrival of syphilis on the scene. Yet syphilis is known to occur simultaneously with and to favour leprosy and its cure is necessary before any improvement of the leprosy can be hoped for.[8] So it can hardly be held that syphilis chased leprosy off the stage. But a reasonable explanation of this curious association is as follows: We know that psoriasis, leucoderma, *lupus vulgaris,* rodent ulcer and other chronic skin diseases were confounded with leprosy from the time of Moses up to and including the days of mediæval leper houses and that the apparent number of lepers was thus increased. After the advent of a new plague which had some features in common with cases of true and false leprosy, most of the latter and no doubt some of the former were in turn confounded with syphilis. So that the suddenness of the decline was probably more apparent than real.

Rogers and Muir, while rightly dismissing the theory of hereditary transmission of leprosy, seem to me to make an error in judgement in dismissing the influence of hereditary susceptibility upon the incidence of the disease and that in a few airy words spoken *ex cathedra.* For example they say:

> Hereditary predisposition of (to) leprosy was believed by Virchow and Liveing who did not believe the disease itself to be hereditary, as it is so exceedingly rarely if ever congenital, and Solano of Columbia later supported this view in favour of which, however, no conclusive evidence appears on record.

Conclusive is a hard word, but suggestive evidence abounds in the authors' own book which refers often to leper families and to the comparative infrequence of infection of Europeans even when in close contact with lepers.[1]

Indeed much more suggestive evidence is afforded in the book for the hereditary tendency supported by Virchow and Liveing (goodly names surely) than for the theory supported by the authors.

Hutchinson also ridiculed the supposed efficacy of the segregation of lepers, but then his obsession by his fish theory may have warped his judgement for once, yet the opinion of so great a man must be given due weight and the addition of his name to that of Virchow must add extra force to the argument of Virchow and Liveing.

Another statement made in the book is:

> Some have explained the disappearance of leprosy from Europe as being due to the immunity acquired by European races during the Middle Ages. In our opinion this cannot be so as in India and other Eastern countries Europeans exposed to infection are as liable to acquire the disease as are the natives of the country.[1]

As such a statement is directly contradictory to the accepted theory of the disappearance of a disease, something more than an *ex cathedra* statement is required and indeed in the book itself figures are available to disprove this statement.

In coming to this conclusion Rogers and Muir seem entirely to neglect consideration of the influence of a tropical climate, hardship, alcoholism, syphilis, malaria and other factors which will act powerfully to break down the relative insusceptibility of Europeans. Take for example the authors' own figures for New Caledonia:[1]

> The reported rates (in the native population) were two hundred per mille in 1899 (the maximum) . . . . in 1914 (Leboeuf) the rates had fallen to 26·6 per mille in the natives and was 12·4 per mille among the Europeans, being twice as high in the European convicts as in the free population.

Again:

> But among certain classes of convicts and galley slaves who have intimate daily relations with the indigenous Canaques, leprosy prevails greatly.[1]

Does it not seem just to conclude that French convicts whose lot is notoriously hard, a class already debauched by syphilis, alcohol and general excess, subjected to the hardships of galley slaves, weakened by a tropical climate, would have their resistance to infection with leprosy lowered and would fall victims to the disease in unusual numbers, especially when exposed to a virulent infection coming from a highly susceptible race of natives?

The authors admit this when they say that the incidence was twice as great among the convicts as in the free whites. Even then the highest incidence was 12·4‰ in the whites as against the highest rate of 200‰ among the natives.

If it be necessary to multiply the evidence against the conclusion come to by the authors, let us regard another extract from their book:

> A Government enquiry (in Natal) in 1886 revealed over 100 cases arising from the two imported 43 years before, while in 1895 one hundred and thirty-two additional cases had been reported, making over 200 in all, every one a native.[1]

(This in Natal where most European households are served by native staffs. Note also that here in Australia we have sixty-six white lepers in six million inhabitants and one hundred and twenty-nine coloured lepers in a coloured population of less than one hundred thousand.)

It seems to me impossible that Rogers and Muir on reflection can or will adhere to their statement that in India *et cetera* Europeans, when exposed to infection, are just as susceptible as natives. Even under the worst imaginable conditions of French convict life in a tropical climate and with a highly virulent infection the maximum incidence in Europeans was only one-sixteenth of the maximum in the native race. And if this contention is to be given up and the comparative insusceptibility of Europeans admitted, must not this insusceptibility be taken into consideration in framing regulations for the control of leprosy in a country of temperate and dry climate inhabited almost exclusively by whites? How much greater will be the resistance of Europeans in Australia, only ordinarily syphilized and alcoholized, living free in the best civilized conditions, than in the case of the French convict in New Caledonia, tropical, moist and with a large native population of great susceptibility?

Rogers and Muir admit and Muir insists upon the great influence of general nutrition, quality of housing and cleanliness of habit as influencing the liability to infection and capacity to overcome infection with leprosy. Probably nowhere in the world are such factors so favourable in all classes of society as in Australia.

To return to my story of the Lister Institute and of the discomfiture of the optimists who believed in the efficiency of antituberculous measures, I wish to draw again the parallel between tuberculosis and leprosy.

Tuberculosis is declining because the susceptible stock is being killed off and prevented by illness, short life and poverty from begetting as many children to inherit this susceptibility as they otherwise would and the tubercle bacillus from passage perforce through more and more resistant individuals is losing its virulence. The same explanation, I think, must be given for the decline of leprosy, a sister disease caused by a twin organism.

The process of development of racial insusceptibility to a given disease is well described in Archdall Reid's "Laws of Heredity"[9] in which tuberculosis and leprosy are specifically mentioned as examples of the law that:

> The sufferings of races have not altered them at all, only the elimination of the unfittest has altered them.[9]

Again:

> It is hardly necessary to quote evidence that races are immune to diseases in proportion to their past experience of them.[10]

To this I think must be added that some races are by nature more resistant than others, but only become immune for practical purposes by the weeding out process of long continued racial infection.

That the immunity is, however, never complete must be admitted and when by reason of passage through a hitherto unattacked and highly susceptible race the leprosy bacillus has developed greatly increased virulence, the organism may be able to overcome the resistance of some at least of an otherwise practically immune race.[1]

But when, as in Australia with its dry temperate climate (except in the north), its high standard of housing and living, its population of highly resistant race, the practical absence of a native population, conditions so unfavourable to leprosy exist, is it not time that the almost penal regulations were reviewed? And when in addition it is considered that new methods of treatment are available, means that should afford in Europeans an excellent chance of cure (55% or more in native races)[1] or at least of clearance of infective lesions, it becomes a matter for very serious consideration whether the internment of lepers of white race at least is not actually harmful. The temptation for patients and relatives to conceal the disease under existing regulations for incarceration of lepers may expose the immediate associates of the patient to infection for a much longer period than would be the case if the patient could be treated without internment, as he can be and is treated with success in countries where segregation is not compulsory.

Internment of lepers is unknown in Great Britain, France, United States (open to correction and control) and is not compulsory even in Scandinavia. In some European countries segregation in their own homes is applied. Throughout Europe cases arise sporadically or are imported. But treatment of hopeful nature is available and notification and inspection of the conditions under which patients live, generally soon eradicates a focus. Even in India it is found that patients now come for treatment at an earlier stage than that at which they were previously discovered, because the value of the treatment is recognized even by the natives. But if there were fear of internment regulations, the story would be different.

I would suggest that the only places where internment of lepers might be useful, are those in which it is impracticable by reason of the huge number of natives affected and that the only places where it is practicable are those, like Australia, in which it is quite unnecessary and may be actually harmful.

That notification and the fullest facilities for treatment, regular inspection of patients and review of the means of treatment adopted and its results, the provision of hospital accommodation for indigent and ignorant patients and those who desire it or who have open and acutely infective lesions, should be maintained, I heartily agree. Even now numbers of patients who have been infected, are after a period of treatment and subsequent observation discharged on licence, but no provision is made for subsequent treatment. And I shrewdly suspect that the authorities do not wish to recognize the only slightly infective forms, such as anæsthetic leprosy, and I know that they will overlook demonstrable physical signs in order to escape the necessity of interning such a patient and I sympathize with them. Though at first it is an unpleasant shock to have one's facts contradicted, however tolerant one may be towards a difference in the interpretation of those facts, still it is impossible not to agree that this is preferable to the internment of a harmless anæsthetic leper. But it is a sign of the necessity for revising the regulation and control of the disease.

I also know that the authorities will only recognize the case as one of leprosy, if the bacilli are demonstrated to them and that sometimes at least the period during which they retain the suspect, is too short and the repetition and scope of their own bacteriological examinations too confined to prove the absence of leprosy.

I wish to ask you on the evidence of the cases I will put before you, what value can be placed on the failure to find bacilli in European patients and also how far the pathology given in textbooks, no doubt gleaned from cases occurring in susceptible races, is misleading when dealing with cases occurring in Europeans?

From the case of Miss R. and that of H.C.P. one can judge how easily a case can be missed and how misleading may be the pathological findings and how prolonged and widespread must be the search for bacilli. From Miss R.'s case it can be judged how long a time can elapse between the appearance of lesions and the discovery of the disease even with the most expert pathological aid.

From consideration of them all we can surmise that some cases are never diagnosed and we can suppose that the disease may become arrested spontaneously in a certain number of instances. We know that in the anæsthetic form the disease endures a very long time, that the resistance is high, that the infection dies out spontaneously in some cases, but leaves the patient more or less crippled. Is it not reasonable to suppose that the infection in some cases dies out earlier than this and leaves less crippling from nervous damage?

All similar diseases behave in this way. Tuberculosis, a twin disease, though it kills many and damages more, not infrequently dies out without leaving any permanent damage of note.

Therefore it seems eminently reasonable to believe that mild and abortive cases of leprosy occur in the community. This belief is rendered all the more strong when we consider the source of the sporadic cases of leprosy that crop up now and then, for example in the north coast district of New South Wales. Most of these patients come from scattered centres, have no acquaintance with previous patients and cannot be traced to a previously known case of leprosy. And if we do not admit mild and unrecognized, but temporarily infective cases of leprosy which may remain quiescent or even undergo spontaneous cure, we can explain the occurrence of sporadic cases of developed leprosy only by suggesting that there must be an appreciable number of other developed cases of leprosy, unrecognized in a well settled community, well served by a large number of well trained medical men, an alternative which seems to me much harder to believe.

Dr. G. H. Taylor in the *Australasian Medical Gazette* of January 20, 1896, published the results of a painstaking and tedious examination of all the patients admitted to the Coast Hospital, Sydney, for all and sundry reasons, with a view to determining how many, if any, showed any signs of leprosy hitherto unsuspected. He relates that in approximately thirty patients he discovered some thickening and tenderness of the ulnar or peroneal nerves with occasional slight terminal anæsthesia and that in three he discovered eruptions of bullæ on the hands and feet. He concluded that the majority of these patients, from whom possible traumatic damage to the nerve had been excluded, as had all other known nerve diseases, were suffering from early, mild or abortive forms of leprosy, or that they were afflicted by a disease not yet recognized.

In *post mortem* examinations he was able positively to recognize in three instances Hansen's bacilli in sufficient numbers to warrant the *post mortem* diagnosis of leprosy, while in two he was able to find acid-fast bacilli, but in insufficient numbers to warrant a differentiation from tubercle bacilli.

The publication of this report did not result, as might be expected nowadays, in a commendation for valuable work done, but in the administration of an official censure, because Dr. Taylor had not communicated his suspicions (in the case of T.O'R. especially) to the President of the Board of Health (see Appendix B).

Certainly the reading of the official correspondence and reports relating to the matter conveys the impression that a definite effort was made to put Dr. Taylor in the wrong and quite possibly youthful lack of tact on the part of the latter made his case worse by inducing injudicious management of the situation. But the strong impression remains and a suspicion is bred that Dr. Taylor was probably right in his fear that he was to be deprived of the credit of his long and tedious research.

But even if it be admitted that Dr. Taylor was technically to blame, the results of his breach of

discipline were so valuable that it is a great misfortune that the fault was not condoned in appreciation of his results and that the authorities then in power did not encourage Dr. Taylor to further his efforts and clinch his proofs by further research, rather than side track him into the routine of the duties of Railway Medical Officer.

As nearly all the principals are now dead and Dr. Taylor has retired from his Government position, no harm can come from the mention of this matter and a tardy appreciation of his efforts by the members of his profession at present in active practice may to some slight degree soften his disappointment in the loss of his opportunity to prosecute an interesting and valuable research.

That his work is not altogether unappreciated is shown by the fact that THE MEDICAL JOURNAL OF AUSTRALIA in its issue of April 28, 1923, and of July 11, 1925, refers to the value and significance of Dr. Taylor's work and *The Lancet* of September 19, 1925, referring to the notices in THE MEDICAL JOURNAL OF AUSTRALIA, points out that the conception of leprosy as a necessarily progressive and inevitably fatal disease, as held by popular and to a great extent by medical opinion, is not necessarily founded on fact. The journal then shows that Dr. Taylor's work preceded and foreshadowed that of Dr. Muir[8] or rather that the work of the latter certainly confirmed much of Dr. Taylor's work by demonstrating leprosy bacilli in many persons who do not manifest leprous lesions and in others who appear to recover spontaneously.[8] [11] The above is a fairly close reproduction of the actual wording of *The Lancet's* report and must be very gratifying even so late in the day to Dr. Taylor in his retirement. Support for this contention is afforded by the descriptions of *formes frustes* in Brittany by de Galatz[12] and the tuberculoid forms of Jadassohn about which Dr. Tebbutt will make particular mention.[13]

It must be remembered that in addition to the cases in which bacilli are found in unsuspected persons, there are others who, though suspected, cannot be made to comply with the requirements of official diagnosis, in that the bacilli cannot be demonstrated even after prolonged search during life.

For the purposes of this paper, too, Dr. Taylor's work is additionally valuable, as it supports, as indeed it really inspired the hypothesis that there exists as a focus of infection a number of undiscovered, unsuspected, practically undiagnosable cases of leprosy, abortive, self-determined, undergoing spontaneous cure or subsiding ultimately into permanent quiescence, but affording the risk, unavoidable by any official measures, of infecting with the disease the occasional "throw back" European with a susceptibility to leprosy. If this be the case and it seems well recognized now, thanks to Taylor's and to Muir's work, is it not time that more elasticity should be allowed in the management of revealed lepers of European blood in countries like ours? Under reasonable supervision and adequate modern treatment a far greater measure of liberty and comfort would seem to most of us likely to result in no palpable if indeed in any real increase in the risk of infection of the population of our country.

Just in parenthesis at this point may I quote from the official correspondence of the Taylor incident a portion of a letter written at the time by Dr. Taylor: "Each time I feel that I have been instrumental in admitting a human being to the most miserable existence I can conceive of, *i.e.*, life in the Lazarette."

This opinion, held by the Medical Officer at that time in charge of the Lazaret, is also undoubtedly held today by many other medical men and, of course, by the patient who is threatened with incarceration in that institution.

This brings about the anomaly of medical men refusing to accept a diagnosis of leprosy on mere physical signs without demonstration of bacilli which, I hope to show, is at times exceedingly difficult to achieve even in cases in the end definitely proved to be leprous and not only leprous, but nodular. These practitioners therefore do not report cases unless they are compelled to do so by the discovery of the bacillus. In this attitude the practitioner is confirmed by the official refusal to accept the diagnosis without the demonstration of bacilli.

This estimation of the lot of the discovered leper also brings it about that the patient, his suspicion having once been aroused of danger of internment and having once had his case found as "not proven" by reason of failure to find the bacillus, is off like a deer that feels the sting of a grazing bullet. After refusing to allow the excision for examination of a cutaneous nerve and in his effort to evade internment, he evades treatment as well and remains a risk to his fellows. Of these two undesirable effects brought about by the existing system of internment I have had conclusive personal experience.

I wish to bring under the notice of this meeting and of the medical profession generally, but especially of the authorities controlling the disease, the very important bearing which such happenings have upon the risk to the community and to ask all concerned if it is not more than questionable whether the existing regulations are beneficial and whether their very severity does not only achieve the defeat of their real object by favouring rather than hindering the spread of the disease, at any rate in a community such as ours, in a country such as Australia.

Since writing the above my attention has been drawn by Dr. Tebbutt to a letter written by Dr. Cook[10] in THE MEDICAL JOURNAL OF AUSTRALIA, September 27, 1924, an article which I had unaccountably missed. In this communication Dr. Cook points out not only the fallacy of insisting upon a positive bacteriological finding for official acceptance of the diagnosis, but the dangers of failing to provide treatment for discharged or doubtful cases, the temptation for the practitioner to refrain from notifying and for the patient to

evade treatment owing to fear of internment along with natives and Chinese. A believer in the theory that the leper regulations in the Middle Ages together with the improvement in hygiene and conditions of life caused the decline in the leprosy incidence, he nevertheless has forestalled me in pointing out the faults of the system of internment of lepers in Australia.

I wish, however, to point out that improvement in hygienic conditions of housing and life cannot rationally be dated back to the fourteenth, fifteenth and sixteenth centuries, the ages of the decline of leprosy following upon the Crusades and the period of the wars with France and Scotland, the Wars of the Roses and including the visitation of the "Black Death." It was not really until the time the Tudors or more especially until Elizabethan times that there really occurred improvement in hygiene and nutrition to a degree likely to check the spread of leprosy. But by this time the disease was already practically extinct. In France, too, a study of the deplorable conditions of life of the peasants right up to the time of the Revolution will not permit the retention of the hypothesis that hygienic and nutritional improvement caused the disappearance of leprosy, since the latter long ere that time had become extinct in France. Nevertheless, I am grateful to Dr. Cook for the support he gives to my contention that there are many disadvantages and fallacies in the existing regulations for the control of leprosy.

At any rate if the possibility of the existence of mild and abortive cases, as suggested by Taylor and Muir, Jadassohn and de Galatz, be borne in mind and if it be admitted, as it must be, that most patients with leprosy who ultimately find their way into the internment lazaret, have been for years infected before internment, for months and years a source of infection to others; if we admit, as we must, the possibility of missed diagnosis, if it be conceded that we are a naturally resistant and a relatively immune race, that by far the greater part of our population is outside the bounds of a climate favourable to leprosy and that our conditions of life and housing are very unfavourable to this disease, that a hope of cure will induce more patients to come into the open than any restrictive legislation, if these or even most of them be admitted, what is the rhyme, reason or justice in continuing the existing practice and condemning fellow countrymen to an indeterminate incarceration, to a fate which they fear with some reason to be worse than that reserved for a convicted burglar or in New South Wales at any rate even for a convicted murderer?

I also maintain that the standards adopted for the discovery of leprosy are too low, if it be seriously believed necessary to intern lepers for the sake of public safety. And if it be not seriously believed, why intern any?

Diagnostic Value of Findings.

Now let us examine how valuable are these tests for acceptance of a diagnosis of leprosy.

(i.) In regard to bacilli in the nasal secretion, of the last four (or five) European patients (two with nodular and two with anæsthetic leprosy) whom I have seen, none yielded bacilli on frequent examination of nasal mucus. (This, I think, is a late manifestation in most cases.)

(ii.) In regard to bacilli in the serum expressed from ulcerated or scarified nodules, in two patients with the nodular form seen during the last ten years no bacilli could be obtained by this means, yet both were ultimately definitely proved to be leprous.

(iii.) In regard to bacilli in sections of an excised nodule, in two nodular cases during the last ten years no bacilli were found even though serial sections were stained and searched by many examiners. Yet both were proved ultimately to be leprous. In one of these patients, H.C.P., the serial sections were stained by both hot and cold methods and examined exhaustively by a competent pathologist and his staff, as well as by myself and an experienced resident and not a single bacillus could be found. In addition to this sections from this patient were examined by the Townsville School of Tropical Medicine and by Dr. Dew in Melbourne and no one could find a bacillus. Moreover, in both cases on account of the giant cell systems the strong and ever stronger finding of the pathological departments was "tuberculous—must be tuberculous." Yet textbook authority can be freely quoted to show that giant cells do not occur in leprosy.[14] The bacilli were ultimately found in a cutaneous nerve.

In the second patient, Miss R., nine years ago a similar picture was obtained in sections from a nodule and no bacilli could be found there, but no nerve was excised. I wonder now if we had thought of it and had obtained a piece of cutaneous nerve, whether we should have found the bacilli.

Professor Welsh saw this patient with me and examined the sections in his laboratory, where at the moment I was doing some work on microscopical appearance of incipient rodent tumours. We concluded on the giant cell systems that this must be an aberrant case of tuberculosis of the skin, but four years later the patient showed up in Brisbane as definitely leprous, so we were confounded before our fellows and humbled in the eyes of the people. It did not hurt so much in such good company, but both the Professor and I remember the lesson well. Hence my obstinate refusal to accept the "probably tuberculous" finding repeated over and over again on histo-pathological evidence in the case of H.C.P.

(iv.) In regard to bacilli in the blood, I have specimens of leprosy bacilli in the blood slides which I obtained in Paris at the Pasteur Institute in 1908, but the patient must be in a febrile stage or the search is fruitless. I could not catch my patients in a febrile stage, though I kept them weeks in hospital until the superintendent became so restive that I had to transfer one to the Coast Hospital for further observation. Thence he was

discharged after a few days, because they could not find bacilli by the ordinary examinations. (I could and did tell them that.) At any rate the patient, Private B., on my subsequent demonstration to the Repatriation Department of the existence of the physical signs of anæsthetic leprosy got a two-third pension, but is officially at least not a leper nor is he having any treatment.

(v.) In regard to bacilli in the nerve leading to anæsthetic area, this is not an official test, but is due to a suggestion for which I am indebted to Dr. Dew. I wish publicly to convey my sincere thanks for the great help he gave and interest he took in a very difficult case.

This, I think, may prove to be the most dependable of all examinations and should be done much more frequently. It is not insisted upon by the authorities nor relished by the patient or by the surgeon who has to be asked to obtain the specimen. In the one case (nodular H.C.P.) in which it was done, it provided proof and revealed bacilli when no bacilli could be demonstrated in nasal secretion, serum from scarified lesions or in sections of excised nodules and lymphatic glands. Moreover, besides the bacilli this nerve manifested another feature which I think must be characteristic, namely, the presence of giant cells in the substance of the nerve in between the nerve bundles. I have not heard of giant cells being found in a nerve trunk in any other disease. (For demonstration of sections of nerve and of nodules I must refer to Dr. Tebbutt's paper.)

The consideration of these cases and of this evidence goes to show that the standard of diagnosis is highly unsatisfactory, if it is earnestly believed that internment of lepers is necessary for the sake of public safety.

Two other patients (Private B. mentioned above and Mrs. J.) were purely anæsthetic and from the clinical point of view certainly lepers, pathologically unprovable, since both refused to have a cutaneous nerve excised. Both are at liberty now and justly so, but both are lepers none the less, anæsthetic to the middle of the forearm and calf with thickened ulnar nerves and anæsthetic patches elsewhere as well. Both were demonstrated to other members of the staff of the Royal Prince Alfred Hospital and to numerous students, one to a staff meeting of the hospital. This is the patient Private B., whose physical signs, then demonstrated, were denied by the authorities as already mentioned.

Of the two patients with nodular leprosy mentioned (I am not now speaking of patients with leonine facies and advanced unmistakable leprosy) one, Miss R., escaped diagnosis for four years and the other, H.C.P., would have escaped diagnosis except that the clinical signs and lesions were inconsistent with tuberculosis as strongly suggested by the histo-pathology and would also have gone untreated for years, unless we had taken Dr. Dew's advice and excised a cutaneous nerve for examination.

So much for the accepted standards of diagnosis as required under existing regulations.

Syphilis and tuberculosis of the skin are the two main diseases with which leprosy is likely to be confused. Now long association with syphilis in large numbers of patients at the Royal Prince Alfred Hospital clinic, one of the largest and the oldest evening clinics in Australia, and fifteen years charge of the Skin Department of the same hospital, have made me fairly quick to recognize syphilitic and tuberculous manifestations in the skin. In view of recent events, however, I am convinced that I have missed cases of leprosy on account of the character of the pathological findings. I know I missed the diagnosis in one patient (Miss R.) on the misconception that bacilli must be numerous in nodular lesions and I would have missed the diagnosis in the last patient (H.C.P.) if it had not been for my obstinate belief in the value of positive clinical evidence as against absence of pathological findings. But I ask myself whether I have not missed many more. I am closely watching another patient now, Mrs. S. (see Appendix A).

If this be so, how often are cases missed when patients are examined by medical men whose limited opportunities do not allow them the same advantage in differentiating from leprous lesions those of syphilis and tuberculosis. In support of this hypothesis that there exist cases which by errors in diagnosis and too great reliance on the bacteriological findings go undiscovered, there is, besides the suggestion that such cases must exist to provide the means of infection for those sporadic cases which are ultimately recognized, the weighty evidence produced years ago by Dr. Taylor and recently by Dr. Muir[11] (see Appendix B).

In any case Dr. Taylor's finding makes it more than ever likely that mild and unrecognized infections with leprosy occur not so infrequently in European communities as would be expected in a population highly but not completely immunized.

We all know in our hearts that the risk to the community of allowing full liberty to all the lepers in Australia, including those who are interned, even without the advantage of modern treatment, is as a drop in the ocean compared with the risk of leaving unsewered large sections of our capital cities or of leaving our city milk supplies no better than they are, not to speak of many other dangers, such as arise from the infectivity of tuberculosis, syphilis and so forth.

Verily do we strain at a gnat and swallow a camel.

By way of comparison with our system of controlling leprosy, let us regard what Rogers and Muir, strong adherents of the system of segregation, publish as their conception of the ideal methods of prophylaxis against leprosy on pages 126 and 127 of their book.

The ideal methods of prophylaxis against leprosy according to them are as follows:

1. Compulsory notification of every case of leprosy immediately it is recognized, by both medical men and laymen, under legal penalties for omission.

2. Examination of each notified case by an experienced medical board, with the aid of microscopical examinations to confirm the diagnosis, and to decide if the patient is in an infective stage.

3. The immediate isolation—best in a special institution such as a leper colony or hospital—of every case from which lepra bacilli are likely to escape from either the nose, throat or skin, the patient being detained until free from all signs of the disease for not less than one year, during which repeated bacteriological tests shall have proved negative.

4. Where home isolation is allowed, only patients with no discharge of lepra bacilli from their skins and nasal mucous membranes shall be permitted to remain in their own houses, when their circumstances satisfy the medical inspector that others living in the house incur no danger and no children or adolescents are residing there.

5. Separation of the sexes and the prohibition of marriages of lepers with healthy persons, or with each other, unless the male submits first to the simple operation of sterilization to prevent the procreation of children or the woman is beyond the childbearing age.

6. The separation of all healthy children of lepers from their parents from birth.

7. When a leper is found, all house contacts and close associates should be examined at once, and every three to six months up to five years, to enable new infections to be detected and treated at the earliest moment.

8. All immigrants arriving with leprosy or developing the disease within its full incubation period of five to ten years, should be repatriated.

The following is a translation of the official text of resolutions of the international conference on leprosy assembled at Strasburg in 1923.

The Third International Scientific Conference on Leprosy maintains the principles of organization of the campaign against leprosy admitted by the preceding conferences and adopts the following resolutions:

1. The legislative measures with respect to the campaign against leprosy should differ according to the countries to which they are applied, but in all cases it is necessary to prohibit the immigration of leprous foreigners.

2. In countries where leprosy is uncommon isolation in a hospital or at home if this is possible, is recommended, such as is practised in Norway.

3. In the centres of endemic leprosy, isolation is necessary. (*a*) This isolation should be humanitarian and permit the leper to live in proximity to his family if this measure is compatible with efficient treatment. (*b*) In the case of indigent patients, nomads or vagabonds and in a general way of people who cannot be isolated in their homes, isolation shall be carried out and the most efficient treatment will be administered in a hospital, a sanatorium or agricultural colony according to the case and the country. (*c*) It is to be recommended that the children of lepers be separated from their parents from birth and be maintained under observation.

4. The members of the families of lepers should be subjected to periodic examination.

5. It is necessary to make the public understand that leprosy is a contagious disease. (Quite superfluous in Australia.)

6. It remains to prohibit lepers from following occupations which allow them to spread infection, but in such case society is in duty bound to realize that this prohibition imposes upon it the obligation of assisting the patient and those of whom he is the support.

Histo-Pathology.

I am, of course, leaving Dr. Tebbutt to discuss in detail the histo-pathology of leprosy as manifested in H.C.P., Miss R. and Mrs. S. and G.T. (lazaret) and as differing from textbook pathology. There are, however, a few main points which I wish to emphasize:

(i.) The comparative infrequence of bacilli in at least some early cases as compared with the open lesions of late cases, especially in coloured patients, which probably provide the bulk of the pathological material of textbook writers.

(ii.) The frequence of giant cells in spite of the fact that some textbooks deny altogether the existence of giant cells and the rest declare them to be rare.[15]

In both H.C.P.'s and Miss R.'s lesions giant cells are extremely numerous and the most striking feature of the histo-pathological picture. Is it possible and I suggest that it is so, that these giant cells are the expression of the higher resistance of the European as compared with the coloured patient?[13]

I should like to draw attention to the very unusual picture of giant cells in the substance of a nerve which I am inclined to believe is not recorded in the case of any other disease and if so, must be considered as strongly suggestive if not pathognomonic of leprosy.

Another minor point is the absence of so-called lepra cells from these sections. Further consideration of these points I wish to leave to Dr. Tebbutt.

Diagnosis.

I do not wish to speak of advanced cases in which with the leonine facies, hairless brows and ulcerated nodules, the diagnosis obtrudes itself, but of the early nodular cases, such as those of H.C.P., Miss R., G.T. (Lazaret), all proved cases; Mrs. S. (doubtful), Chinese girl (doubtful) and others of this kind.

In all these cases the first impression is that usually experienced on inspection of a syphilitic lesion. The colour and shape of the lesions were strongly suggestive of syphilis in the middle or late secondary stage, though the consistence of the infiltration was softer than that of a syphilitic and more reminiscent of a tuberculous lesion. In almost all these cases syphilis headed the list in the differential diagnosis, but was finally excluded by repeated failure to react to the Wassermann test and complete failure on the part of the lesions to respond to antisyphilitic treatment.

In the meantime discovery of anæsthesia, only partial in most and in H.C.P. slight thickening together with insensitiveness of the ulnar nerves, aroused suspicion. The next step was bacteriological examination of nasal mucus, of serum expressed from scarified nodules and the excision of one or more nodules. In the first of the recent series, H.C.P., the false scent of tuberculosis then crossed the trail. But made wary by the disastrous results of yielding to this suggestion in the case of Miss R. years before, I was not to be drawn off the trail. To justify my refusal to accept in this case the histo-pathological diagnosis of tuberculosis, I felt

bound to exclude this disease. So von Pirquet tests were made without result, a series of old tuberculin injections was given and a guinea pig was inoculated with portion of an excised nodule without yielding evidence of tuberculosis. This done, in this man's case at any rate, the only disease consistent with the clinical picture which remained, was leprosy, but we could not clinch the diagnosis, search as we would. A lymphatic gland, slightly enlarged in the neighbourhood of active nodules, was excised and manifested the same giant cell and granuloma formation but no bacilli. Finally Dr. Dew suggested the excision of a cutaneous nerve leading to an anæsthetic area. This was done, giant cells as usual were found, but in addition a few but unmistakable acid-fast bacilli. As may be imagined this search took many weeks and a host of sceptics had to be answered. But the final success was worth it all, since it established the principles that even in nodular cases the bacilli may be almost undiscoverable and secondly that true giant cells occur in leprous lesions even in a nerve, not to speak of the advantage of securing early treatment for the patient.

Then the succeeding patients, Mrs. S., the Chinese girl and G.T. (Lazaret) were subjected to Wassermann tests; all failed to react. The first two were subjected to tuberculin tests and both failed to react (G.T., not being our patient, was not tested in this way). Mrs. S. was given two doses of "Novarsenobillon" without result and the same will be done for the Chinese girl who first appeared on June 22, 1926. Serum from nodules and nasal mucus were examined for bacilli without success in all three. Snippings of nodules in the case of Mrs. S. contained giant cells and granulomatous formation but no bacilli. Snippings from G.T. contained no giant cells but granulomatous formation with epithelioid cells and no bacilli.[1] Those from the Chinese girl manifested no formation in sections so far examined suggestive of leprosy.

In these last three cases we are at present in a quandary. Mrs. S. has only one lesion on the face with practically no anæsthesia as yet at any rate. G.T. has some anæsthesia of the hands and we suggest the excision of a cutaneous nerve, but he is not in our charge. The Chinese girl has some very doubtful diminution of tactile sensation, but no diminution of pain in fingers of one hand and in her case and that of Mrs. S. we do not know whence to excise a nerve with a reasonable prospect of success. Time alone can show. But in the meantime in each case the picture is consistent only with leprosy, but we cannot notify and fear that both Mrs. S. and the Chinese girl will disappear.

These cases, but especially and completely that of H.C.P., dispel Macleod's doubts about the presence of true giant cells in a case of leprosy uncomplicated with tuberculosis[15] in that in his case giant cells were very numerous, of Langhans's type and tuberculosis was completely excluded by von Pirquet and subcutaneous old tuberculin tests and inoculation of portion of a nodule into a guinea pig, all without result.

But in addition they all show the extreme difficulty of early diagnosis, if we are to be bound to bacteriological corroboration.

My personal opinion is that they all have leprosy except perhaps the Chinese girl, all simulating diseases having been excluded; but how to prove it in the absence of a leprin without losing valuable time for treatment I confess I know not!

Since November, 1925, beside H.C.P. and Mrs. S. and the Chinese girl who are nodular, we have also had two purely anæsthetic patients (Mrs. J. and Private B.), both unacknowledged, but both clinically lepers. This succession is simply the curious freak of fate which so often happens to produce a string of cases of rare disease after a long gap.

In the two anæsthetic patients of course no bacilli can be found; that is nothing remarkable. Both refused to allow the excision of a cutaneous nerve, one I suspect because of the advice of the physician who asked me to see her in consultation. He admits she is not suffering from Morvan's disease or syringomyelia, but calls her condition Raynaud's disease. The other patient was scared by being sent out to the Coast Hospital and has disappeared, though I was willing and anxious to treat him in the Out-patient Department of the Royal Prince Alfred Hospital.

As it appears to me in early cases of nodular type we must first have our suspicion roused by the combination of syphilitic appearance and tuberculous consistence of the lesions or perhaps by a degree of anæsthesia. Then by exclusion of syphilis and tuberculosis we arrive at the clinical diagnosis of leprosy. Next we must purge our minds of the fallacious idea that in all and especially in early cases of leprosy bacilli are numerous and always easily found. A prolonged search lasting many weeks and including the examination of a nerve, may be necessary to clinch the clinical diagnosis by bacteriological method. And we must also discard the doctrine that true giant cells do not occur in leprosy.

In anæsthetic forms the loss of both tactile and painful sensations (including of course the loss of differentiation between heat and cold) and the presence of anæsthetic patches here and there on the body should render reasonably certain the exclusion of syringomyelia (the most dangerous possibility of error, fortunately rare).

Especially is this so if the ulnar or peroneal nerves are thickened and insensitive to the severe pressure between the examiner's fingers and the condyle. If no pins and needles sensation referred to fourth and little finger is experienced by the patient, there is a strong probability of leprosy.

Tabes dorsalis, hysterical anæsthesia, traumatic damage to the nerve *et cetera* should be readily enough excluded.

Suspicion should be commoner, search and examination wider in scope and I think then that diagnosis would be more frequent.

[1] Since this paper was written bacilli have been discovered by Dr. Tebbutt.

Treatment.

The modern use of esters of chaulmoogra oil and hydnocarpus in combination with potassium iodide and antimony seems to afford, especially in early cases, a very good chance of cure.[1] Rogers and Muir say:

> Improved methods of treatment, holding out great hope of eventual recovery, especially in early cases;

and again on page 134:

> To enable all the infections arising from the first case to be detected and cured in the early amenable stages.

In order to obtain such good results, however, Muir insists that it is necessary to provoke mild febrile and local reactions which determine comparatively rapid diminution of leprous lesions. But for this purpose the method of intravenous injection or intramuscular injection at least is necessary and dosage has to be carefully worked out for each individual patient.

For full description of modern treatment see Muir's "Treatment of Leprosy," *Indian Journal of Medical Research*, October, -1923.

The Coast Hospital Lazaret, New South Wales.

A visit was made to the Lazaret at the Coast Hospital, New South Wales. Sixteen patients were seen. The male patients were inspected on June 12, 1926. The impression gained was that the majority of the patients were excellently cared for and wonderfully cheerful in spite of their hard lot. A greater degree of liberty is allowed than was anticipated and the situation of the Lazaret is pleasant and open, even picturesque. The buildings are detached, mostly wooden and could well be treated to a new coat of paint, but the quarters are comfortable and clean.

Dr. Tebbutt and I saw six patients in the clinical room, patients who were active physically and under active treatment, as well as two anæsthetic patients in the last stages, one a Chinese and one a Javanese, bedridden mostly by reason of old age and one patient suffering from an acute nodular leprosy, a Greek, who is not having any medicinal treatment for some religious reason.

The last is really the only patient who manifests any remarkable lesions, the only one who would attract the eye of the layman, the only one who might shock the susceptibilities of even the most fastidious citizen. His appearance more nearly approached that given by illustrations in skin text-books of fifty years ago than I had thought possible in Nature.

Of the six ambulant patients only the Chinese patient and the ex-soldier of the Indian Army would be at all noticeable as abnormal. Of the four British (one Irish, one English and two Australian), three would pass as normal to all but the most searching eye and would not suggest leprosy to any but the initiated.

Discussing the matter with the Superintendent of the Coast Hospital, Dr. Millard, who very kindly gave up a whole morning to us and our inspection, I was impressed with his disbelief in the danger of completely freeing the patients and on the other hand with his suggestion that a refuge for the leper, hounded by his neighbours, was absolutely necessary apart from therapeutic reasons.

It seems that the internment of lepers is largely due to public ignorance rather than to official conservatism. But surely a quiet relaxation possibly under a new *régime* should succeed in freeing those who wish to remain at liberty and who are willing to continue treatment.

The next impression gained was one of disappointment in the nature and as a corollary to this in the results of treatment. Although beneficial results were being obtained, I was not at all satisfied that either the best methods and drugs were being used or that the best results were being realized.

The treatment was being administered largely by mouth, a method by which the nauseating effect of chaulmoogra oil is brought about and this, I understand, is the reason for the Greek developing religious principles. This manner of administration has fallen into disfavour in the best leper clinics.

Also a preparation used for intramuscular injections was chosen largely for its cheapness and is therefore presumably inferior to the more expensive preparations. In reply to a question why "Moogrol" (a high class Burroughs, Wellcome preparation) was not being employed, the reason given for not using it was because it was too expensive.

It seems worth trying this preparation, spoken of with approbation by Rogers and Muir treating patients in India, against the preparation called "E.C.C.O." at present used at the Coast Hospital as a probably finer and more dependable drug.

Again the occurrence of febrile reactions was avoided at the Coast Hospital Lazaret, whereas in Rogers and Muir's book it is expressly stated that the provocation of such febrile reactions is highly desirable from the point of view of obtaining a cure.

To quote Rogers and Muir (page 255, third paragraph): "A severe and alarming reaction was followed by great improvement, which once more illustrates the principle that reactions are necessary in order to obtain much benefit in leprosy" (see also other parts of the book and other authority than that of the authors themselves; also see Muir's "Treatment of Leprosy," *Indian Journal of Medical Research*, October, 1923).

Another missing detail was the use of local infiltration of the obvious lesions with antileprous esters. This method is apparently of great importance and is described on page 264 of Rogers and Muir's book.

Nor is potassium iodide or antimony used as recommended for the production of necessary reactions in patients tolerant of the chaulmoogra or hydnocarpus drugs in the sense of failing to react in a febrile manner and therefore tardy in response to treatment by them.

Altogether I felt there was room for more variation and individuality and imagination in the conduct of the treatment of different patients.

It was admitted that results such as described by Rogers and Muir in Hawaii and elsewhere were not obtained in the Lazaret. But it must also be admitted that the methods followed were not those used in Hawaii, nor those described in "Muir's Treatment of Leprosy"[16] which were expressly designed to provoke moderate febrile reactions with a view to hastening cure.

I wish to make myself quite clear as being a friendly critic, only desirous of suggesting betterment of the lot and outlook of the leper, while greatly appreciating the general care and comparatively pleasant conditions afforded the patients. For example there was only one patient, the Greek with religious principles, who manifested any open lesions. No ulcers or other acutely infective lesions were seen. This speaks volumes for the general care of nutrition and health.

I am informed that conditions at Peel Island are not so satisfactory. There is a population of about fifty lepers on the island, but there is no resident medical officer. It seems, to say the least, unfortunate that such a large institution, containing patients all requiring special treatment, should be visited merely once or twice a week, since under such a *régime* special consideration for individual requirements in therapy cannot be expected and treatment must partake more of the routine character than is usual even in government institutions. Hence the best results cannot be expected.

Recalling the patients seen at the Coast Hospital, several points beside the absence of open lesions impressed me.

One was the syphilitic appearance of the lesions which had spread peripherally, strongly reminiscent of the papulo-serpiginous lesions of the late secondary stage of syphilis, both in form and colour. But in consistence there is a difference, the infiltration being of the pulpy tuberculous character rather than the firm rubbery solidity of syphilitic lesions.

Another was the colour ("bistre brown" of Ashburton Thompson's naming) of the macular lesions—a reddish, rather cayenne-pepper-tinged brown, not seen in other diseases.

The third point was the comparative rarity of gross thickening of the ulnar nerves. Thickening there was in most, but small in degree, hardening in most, tenderness from acute neuritis in one only, but a physical sign which I have not seen described elsewhere was present in all but one. This is that severe pressure upon the ulnar nerve between the fingers of the examiner and the patient's internal condyle (so severe that the snap, as the nerve slips from under the fingers, can be heard by others than the examiner) does not occasion the patient any pins and needles sensation, any "funny bone" feeling, referred down the ulnar side of forearm to the fourth and little fingers. Only two patients experienced this sensation and of these one was only a suspect, though probably suffering from a very early nodular form of the disease (this excepting the man with active neuritis who was too tender to test). I would commend this sign to careful consideration and testing, but strong fingers and brute force should be used, if the patient experiences no inconvenience with moderate pressure. If nothing more than local discomfort is occasioned, no pain or tingling referred to the distribution of the nerve, there is strong evidence of grave impairment of the sensory path in the nerve which hardly occurs in such a large trunk in any other condition except leprosy and trauma. And the latter can be easily excluded.

The following is a brief *résumé* of the patients seen in the Lazaret:

CASE I.—G.T., an Englishman (suspect only), fifty-six years of age, worked on Peel Island for five years; left nine years ago. First lesion appeared six months ago, a patch on left hand on the site of a wound sustained during his work on Peel Island; later patches appeared on right hand and both elbows. Patient is anæsthetic to pain over the greater part of both forearms and hands. His ulnar nerves are not thickened, but are sensitive to firm pressure with pins and needles sensation referred to fourth and little fingers. Lesions are circinate, syphilitic in appearance, the consistence of the infiltration is pulpy. Wassermann test yielded no reaction.

No bacilli were obtained in serum expressed from lightly scarified lesions. (For later pathological findings see Dr. Tebbutt's paper.)

My opinion was strongly in favour of early nodular leprosy.[1]

CASE II.—A Chinese, about forty-five years of age (advanced mixed type) had some well marked deformity and atrophy of extremities. The ulnar nerves were thickened, no pins and needles sensation was produced on pressure.

CASE III.—B., an Irishman, aged sixty-one years, had only two or three indeterminate papules. He is an old tubercular leper at present quiescent. His ulnar nerves are scarcely thickened, but hard and give no pins and needles sensation on pressure.

CASE IV.—A.C.P., aged twenty-nine years, came from Northern Rivers, New South Wales, and was first admitted in 1914 with nodular lesions on cheek now healed. Has some thickened skin on the right knee from which bacilli can still be obtained. His ulnar nerves are hard, not thickened, no pins and needles sensation was produced on pressure.

CASE V.—P., aged forty-one years, a Greek, had huge nodular lesions on the face, hands and forearms; the nerves are thickened but sensitive. He declines treatment.

CASE VI.—A Chinese, says he is fifty-four years old, but looks older. He has been fourteen years in the Lazaret. His old tubercular lesions are healed, but nervous involvement has caused deformity of hands and feet. He refuses to leave bed, though well enough to do so. His ulnar nerves are not thickened.

CASE VII.—A Javanese (a very old man), has been thirty years in the Lazaret. He suffers from anæsthetic leprosy. He has lost fingers and toes. He suffers from senile dementia and has been confined to bed for years. His ulnar nerves were not tested.

CASE VIII.—H.C.P. (my own patient) has nodular lesions on the face and arms, subsiding under treatment. The ulnar nerves are slightly thickened, no pins and needles sensation is produced on pressure.

CASE IX.—An Englishman, over sixty years of age, suffers from active mixed leprosy. He has served in the Indian army. Numerous subcutaneous nodules are present on the legs. The ulnar nerves are very tender and in a state of active neuritis. Apparently his condition is unfavourable.

[1] Fuller details concerning this patient will be given in a subsequent issue by Dr. George R. Hamilton.

Conclusion.

What is the reason that leprosy alone of all the chronic infective diseases is treated in this way by internment and deprivation of almost all civil and human rights? Is it because it is so dangerous to our community? Certainly not! This we can see by the absence of regulation for isolation of lepers in Great Britain, in the United States and in France, where patients with leprosy exist in fairly large numbers (two hundred in France), but are nearly all imported; yet in each country lepers number only a couple of hundred or so among populations of many millions. What is the situation here in Australia among the whites? In Sydney there are six male and seven female revealed lepers, in Queensland forty-one male and ten female lepers, in Victoria none, in South Australia none, in Tasmania none, in Western Australia two—a total of sixty-six white lepers in six million inhabitants. What of it? Whooping cough probably kills more and we all have to die anyhow and nowadays lepers die of old age mostly—hardly any of them from leprosy. No! it is because of the horror inspired by the disease and the ignorance of its appearance, course and possible cure. And this horror, is it a present day horror? Most certainly no! If you wish to confirm my answer, go on your next visit to Sydney and ask Dr. Millard to show you over his Lazaret. If you are looking for sensationalism or horror you will be disappointed! There you will find four or five ordinary looking whites languishing in company with some Asiatics employing their time in gardening, fishing and biting their nails. That is the only horror you will experience.

No! this horror is a curious instance of the survival of race impressions. No doubt the conglomeration of diseases which constituted mediæval leprosy, produced many horrible cases. But imagine the situation if syphilis had existed in the Middle Ages and had been untreated as leprosy was untreated. Would it not have provided many more, much more horrible pictures than leprosy even then presented? As lay evidence see the paragraph from the Sydney *Bulletin*, June 10, 1926:

> Leprosy is not a hideously disfiguring disease as writers, modern as well as ancient, have led people to believe. . . . At a parade of patients in one of the largest lazarettes in the Pacific . . . . only about one in five bore any external signs of the disease and few of these were the monstrosities that a layman would expect to see. Probably the most ghastly of all diseases is tertiary syphilis, with the "gangosa" or *rhinopharingitis ulcerosa* of the north-west Pacific, running a ghastly second.

But we do not intern syphilitics; of course not, we allow them to live and work and spread their infection and fill our hospitals and insane asylums with nothing but mild legislative restriction to become invalid and insane by the thousand. This horror of leprosy is nothing but a survival like superstitious beliefs about passing under a ladder or witchcraft which came down to us from our ancestors and it should be discarded as we have shed others.

If we called leprosy by another name would it not smell much sweeter? Let us call it bacillary granuloma or bacillary neuritis and where, with modern methods of treatment, will the horror go? We should never know where—into the *Ewigkeit*—into the limbo of other superstitions.

And anyhow are we justified on the evidence before us in subjecting the unfortunate leper to penalties over and above those inflicted by fate? In Norway a flat is now produced in the curve and that flat apparently exists here also and would be found all over the Anglo-Saxon world, if figures were available. And can we do any worse by rational treatment and management than by incarceration of revealed lepers? I appeal to you; think of tuberculosis and the risk of infection and death thereby; think of syphilis untreated; let us cleanse our mind of the horror of the name of leprosy. Let us start afresh and originate a clean, unprejudiced and rational *régime* for the management of the problem of leprosy and let our humanity extend liberally to the unfortunate lepers, at any rate to those who are willing and able by reason of their intelligence and education to follow out treatment and to avoid any reasonable risk of infecting their fellows.

We know that leprosy is not nearly as infective or dangerous as tuberculosis or syphilis. Therefore let us treat its victims more lightly than we treat tuberculous and syphilitic patients rather than as now like criminals.

In conclusion I must admit that I have perhaps given too much rein to what my dear old Anglo-Saxon friend, Moran, calls my Celtic passion in handling this subject of leprosy and the regulations governing it.

I have, however, done this purposely, because it seems to me high time that the prevailing practice of handling leprosy patients be fully reviewed in a strong humanitarian spirit. If you have seen the tragedy in a man's eyes and heard the despair in an educated European's voice, not so much at being told he was suffering from leprosy, but at being told that he must go to the Lazaret; if you had heard a man say that but for the hope of cure I was able to hold out to him, he would rather blow out his brains than submit to internment, you would sympathize with my effort to bring the subject up for reconsideration and have the regulations amended and administered uniformly and in the best possible way under the best possible authority. And this I am sure you will do if you realize the pathos, as I hope I have convinced you of the injustice and futility of the fate reserved under the existing regulations for the leper of our own race in this our own country.

Appendix A.

The following are brief histories of patients.

Case of C.E.B.

On February 18, 1924, the patient, aged forty years, came from Darwin. He had large yellowish macules, some several inches wide, scattered over arms, abdomen and back. Sensation was definitely impaired, especially in the middle of each patch and on hands. Ulnar nerves were thickened. He suffered from anæsthetic leprosy. This was confirmed on the first bacteriological examination of

serum from macules at the Board of Health. He was reported and returned to Queensland. The case is of interest on account of unexpected ease in finding bacilli in macular lesions.

Case of Mrs. J.

On March 11, 1926, she complained of losing feeling in hands and feet for nine years. She was thirty-six years of age Fingers became affected first painfully, then with numbness. She could feel only when she pressed hard. The feet later became affected in a similar manner.

Blotches appeared on the face and back three months ago. She burned her fingers while cooking without being conscious of having done so until she saw the damage done. She had three burns on the hands. On examination the skin was atrophic on fingers and toes, the fingers were pointed. Light brown patches were present on chest and back and on the arms to above the elbows and on the legs to above the knees. She was anæsthetic to pain and tactile sensation (apart from deep pressure) was lost on hands and forearms, feet and lower legs and on other areas on the trunk (see Figure I.).

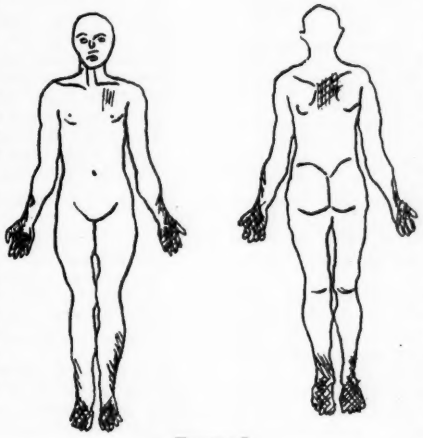

FIGURE I.

No bacteriological confirmation could be obtained.

The patient declined to have cutaneous nerve excised.

Case of Mrs. S.

The patient was an out-patient at the Royal Prince Alfred Hospital. She had a circinate lesion on the cheeks and forehead crossing the eyelids with soft infiltration at the edge, little or no atrophy in centre and no ulceration. The lesion had been present only for eight months. She was sent in as suffering from *lupus erythematosus*. The patient is a Hindoo born in Madras; she went thence to New Caledonia, but has lived continuously in Sydney for thirty years.

The Wassermann test yielded no reaction, two "Novarsenobillon" injections (0·6 gramme on each occasion) caused a ? slight improvement. A snipping was removed from the edge of the lesion; giant cells were found, but no bacilli.

Owing to the patient's colour a von Pirquet test was not done, but the patient was admitted and a series of three old tuberculin injections up to the maximum dose was given without the occurrence of any reaction. Another snipping was taken at the same time, but again no bacilli could be found. We are therefore at an *impasse*, but I am convinced that this is a leper with the first lesion. There is, however, no anæsthesia, no thickening of ulnar nerves which give pins and needles sensation on moderate pressure.

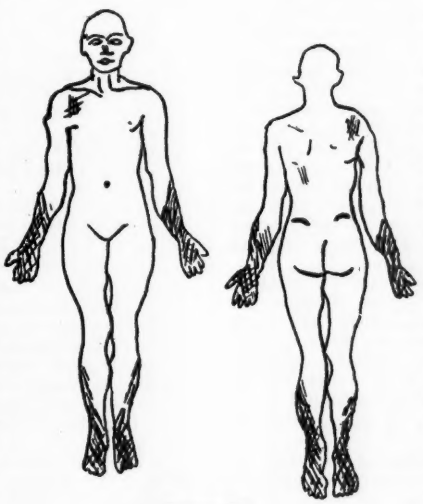

FIGURE II.

We cannot report without bacilli being found and do not wish to scare the patient away by suggesting the diagnosis.

Case of Private B.

The patient was aged fifty years. He was sent by the Repatriation Department on November, 1925, for an opinion as to disability by reason of anæsthesia. He is an oysterman by occupation. He has lived in New South Wales except during the war, when he was in Egypt for some years. While in Egypt he was in hospital with a "dermatitis" which did not weep and passed off. Military histories were not definite, but the condition described is suspicious of prodromal erythemata. Since his return he has found it increasingly difficult and finally impossible to work on account of his inability to feel when groping in oyster beds.

On examination he is a big muscular man, rather stupid and dull. He has anæsthesia of both hands and forearms nearly to the elbow and of both feet and legs nearly to knee (see Figure II.). Several other patches of anæsthesia are present on the right shoulder at back and dulled sensation elsewhere. Forearms manifest white patches with faint atrophic changes. Both ulnar and peroneal nerves are thickened and insensitive to severe pressure.

During his stay in hospital he got an erythematous patch on the buttock, no vesiculation or weeping was present. The patch was not eczematous, but no bacilli could be found in serum expressed from it. The patient had no rise in temperature during the attack.

No bacteriological confirmation could be obtained and the patient declined to have a cutaneous nerve excised.

Case of H.C.P.

On October 28, 1925, the patient was aged forty-one years. He was sent from Mackay (Queensland) with a letter from Dr. Earle Page who had seen him in consultation with Dr. Gallagher and had had a snipping taken. A report on this snipping, examined at the Townsville School of Tropical Medicine, was "probably tubercular" on account of giant cell systems found.

A papular infiltrated rash was present on forehead and body. On lower leg there was a large area with yellow centre and brown edge. All lesions had a syphilitic colour and were distinctly infiltrated. Sensation was distinctly dulled over large patches and little or no pins and needles sensation was present on rolling the ulnar nerve against the condyle of the humerus.

He has lived in Queensland all his life. The condition was almost certainly a Hansen's bacillus infection. He was sent to the Royal Prince Alfred Hospital. His serum was examined by the Wassermann test and no reaction was obtained. Two more snippings were taken, the second from a papule which was actively increasing in size; these manifested giant cell systems, but no bacilli could be found in sections nor could they be discovered in the nasal secretion or in serum from other scarified nodules. A lymphatic gland which was slightly enlarged and tender, was excised with the same result—giant cell systems, but no acid-fast bacilli. This made the Pathological Department strongly inclined to support the Townsville School of Tropical Medicine in its diagnosis of tuberculosis.

Sections were sent to Dr. Dew, who was also unable to find bacilli. After further correspondence Dr. Dew

suggested excision of a cutaneous nerve leading to an anæsthetic area and this was done in the calf. In this nerve giant cells were found in between nerve bundles and some acid-fast bacilli. The patient was admitted to the Lazaret and is now progressing favourably.

Case of Miss R.

On October 8, 1917, the patient, aged twenty years, had a large infiltrated deeply pigmented flat patch on the back and a psoriasiform syphilide on palm and a similar lesion on the forearm.

On the history together with the clinical appearance I was hasty in making a provisional diagnosis of syphilis. A provocative dose of 0·4 gramme "Arsenobenzol" was followed by failure to react to the Wassermann test.

On October 16, 1917, the lesion was reported as improving.

On October 25, 1917, an 0·3 gramme intravenous dose of "Diarsenol" was given.

On November 11, 1917, there was no further improvement. The result of examination of a snipping taken ten days previously was: "Typical tubercular infiltration all along under epithelium and down along skin appendages —lupus type."

A further history of this patient was that the first patch appeared six years ago on the palm of the hand and gradually increased in size. The patch on the arm appeared ten months ago, first causing numbness. The patch on the face appeared eight months ago.

The entry in the laboratory note book at the Pathological Department, University of Sydney (R. 45772, No. 1) was as follows: "Well marked giant cell systems. Tubercular infiltration all along under epithelium and down along hair follicles and sweat glands. Little or no caseation. Epithelium rather thinned and in section absent over one patch (probably torn off in cutting). Ziehl-Neelsen stain— no bacilli found."

Four years later the lesion was proved by discovery of bacilli in Brisbane to be leprous. The patient was interned in Peel Island.

Appendix B.

Dr. Taylor's Patient, T.O'R.

T.O'R., aged seventy-one years, was admitted to the Coast Hospital under the care of Dr. Shaw, the Senior Medical Officer, on May 31, 1895. He was then suffering from an abscess in the right thigh and was discharged, apparently well, on July 1 of the same year. He was readmitted on October 2 and remained until his death on November 9, 1895. At that period there were two assistant medical officers, Dr. F. C. Shaw and Dr. Henry Sinclair Fordyce. "Both of these gentlemen," Dr. Taylor wrote, "had examined all the patients in the Lazaret and were fully aware of my method of examining patients in the various wards of the hospital, having a possible case of leprosy in view. They assisted me in my work and both of them at various periods brought cases under my observation."

Prior to T.O'R.'s admission to the Coast Hospital in May, he was a patient at Prince Alfred Hospital for a few days and between his admission to the Coast Hospital in May and his readmission in October, 1895, he was a patient under the late Dr. Sydney Jamieson for a few days and was discharged with a note to the hospital depôt and readmitted to the Coast Hospital.

Dr. Fordyce's report on this case is instructive. It reads:

> He was admitted into this hospital on May 31, 1895, under my care and was discharged apparently well on July 1, 1895. He was then examined by me in the routine manner. His circulatory and respiratory systems were apparently normal, his urine was normal, there was no eruption of any sort on his body. The only obvious lesion about his person was a chronic abscess on the inner side of the left thigh just above the knee. His knee jerks were present. For a man of his years he seemed exceptionally vigorous. With rest and treatment the abscess healed up and he was discharged. The next time he came in he was under Dr. Shaw's care. I saw him one day, but observed nothing beyond the fact that the abscess had again broken down. Some time after his readmission I heard Dr. Taylor mention him as an interesting case. He transferred him under his own observation and spent several days going over his various symptoms. I went up personally to examine this case and although then having the suspicion of a possible case of leprosy, after careful examination I was not at all convinced that the signs and symptoms pointed to leprosy. Many of the symptoms appeared to be due to senile decay.
>
> Dr. Taylor discussed the case freely with Dr. Shaw and myself, but I never gathered that he had more than a suspicion that it might be leprosy. I have examined patients in the Lazaret, in these there were obvious signs of leprosy, but in the case of T.O'R. there were no obvious external signs of the disease. In my opinion only a person looking out for leprosy could have even suspected it and careful and repeated examinations must have been necessary in his case.

Dr. Shaw's report is in a similar strain and concludes with:

> Of all cases of leprosy I have seen this was certainly by far the least marked. Until the time of his death there was nothing to lead me to suppose he was an undoubted leper. I believe that without the detection of the bacillus of leprosy it would have been impossible to certify him.

A few days prior to his death and when he was apparently in good health Dr. Taylor permitted him to leave the hospital, to visit a friend of his in Sydney. He did not return to the hospital until the following day when he was obviously ill with evidence of acute pleurisy. On the day prior to his death Dr. Taylor expressed serum from one of his eyebrows and therein found leprosy bacilli.

Dr. Taylor wrote as follows:

> I had prior to this discovery reported to the Health Department that he had symptoms suggestive of leprosy and was near to his death. On the day following the discovery from the serum from his eyebrow he died. I therefore knew after his death and at the time of the *post mortem* examination that the condition of his ulnar and peroneal nerves was leprous and at the *post mortem* examination I removed both of his eyebrows, a piece of his spleen, his ulnar and peroneal nerves and one testicle. A very unusual condition was the fact that his eyebrows, in which there was a very slight infiltration, had still the normal amount of hair. It would be expected that the slight leprous infiltration of his eyebrows would be followed by the falling out of the hair in that region.
>
> The summary remarks of the bacteriologist who examined the eyebrows, nerves, spleen and testis taken by me from the body of T.O'R. is of great interest when contrasted with the clinical history of his case. It reads:
>
> It will be observed that leprosy bacilli were found in every tissue examined. Since these comprised skin, nerves, spleen and testis it is safe to assume that they were distributed throughout the whole body and that incalculable numbers were present. The extensive fibrous changes observed in the nerves and especially in the testis showed that the disease had existed for a considerable time.

Dr. Taylor regarded this case as one of great interest. From it he thought it might reasonably be inferred that a majority of cases of early invasion by the bacillus of leprosy must escape observation and that the condition of invasion might subside before there was any external sign of the disease. He held that the method he adopted at the Coast Hospital, of examining as many patients as he could with the assistance of his colleagues with a view to possible recognition of early leprosy, combined with an examination of the spleen and of any nerve condition suggestive of nerve bacillus was the only means which enabled him to write his paper and to form the opinion of leprosy therein expressed.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. H. TEBBUTT.

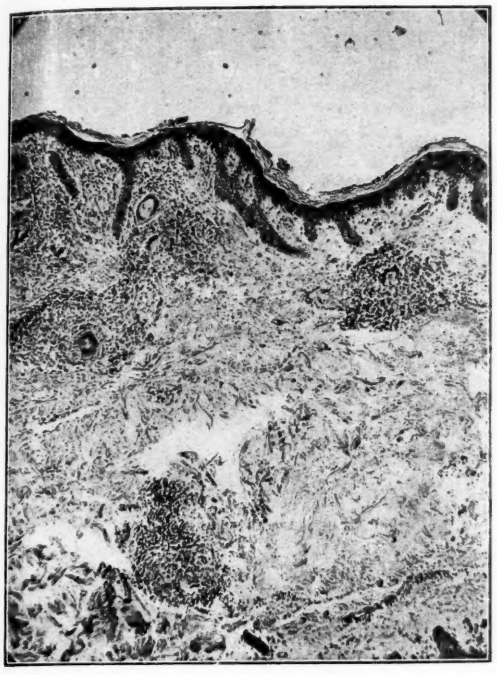

FIGURE I.
Case I., Miss R., Tuberculoid Leprosy. Section of Skin Showing the Infiltration and Giant Cells. × 65. Hæmatoxylin and Eosin.

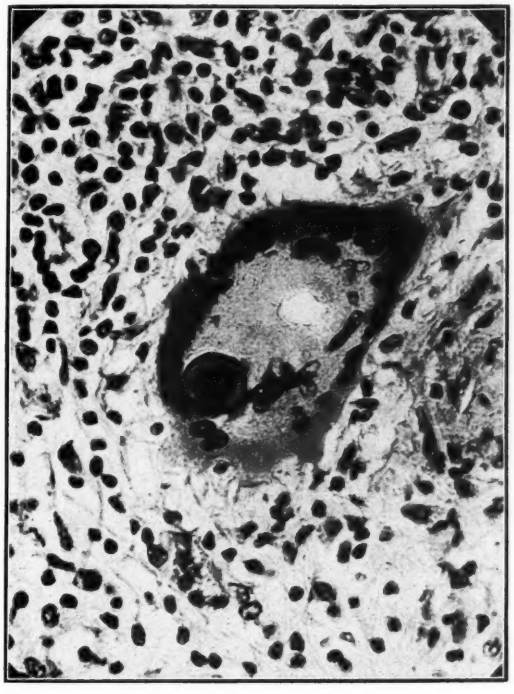

FIGURE II.
Case I., Miss R., Tuberculoid Leprosy. A Follicle with Giant Cell Containing Peculiar Foreign Body. × 500. Hæmatoxylin and Eosin.

FIGURE III.
Case II., H.C.P., Tuberculoid Leprosy. Section of Skin Showing the Infiltration. Two giant cells are seen above the centre. × 65. Hæmatoxylin and Eosin.

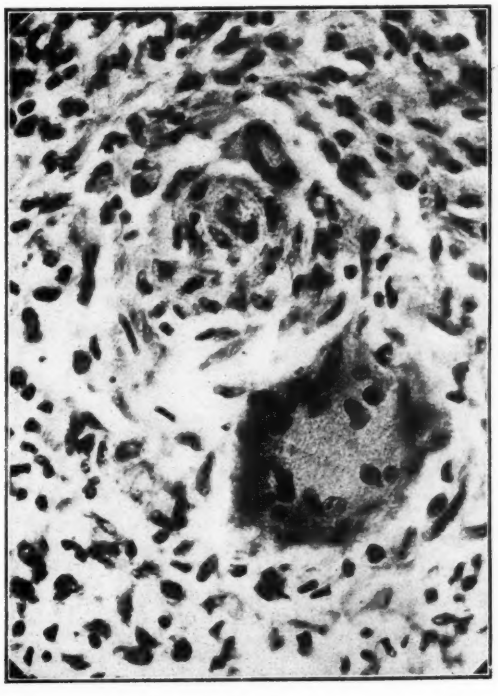

FIGURE IV.
Case II., H.C.P., Tuberculoid Leprosy. A Follicle with Giant Cell. × 500. Hæmatoxylin and Eosin.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. H. TEBBUTT.

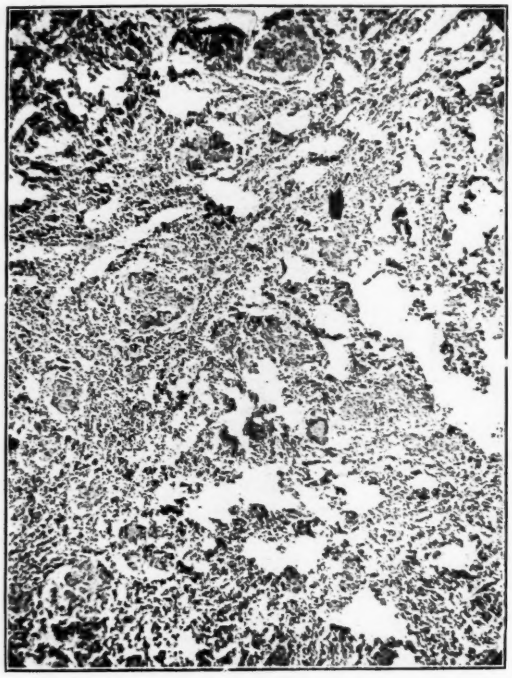

FIGURE V.
Case II., H.C.P., Tuberculoid Leprosy. Submental Lymph Gland. Collections of epitheloid cells and giant cells can be seen particularly at bottom centre and left centre. × 65. Hæmatoxylin and Eosin.

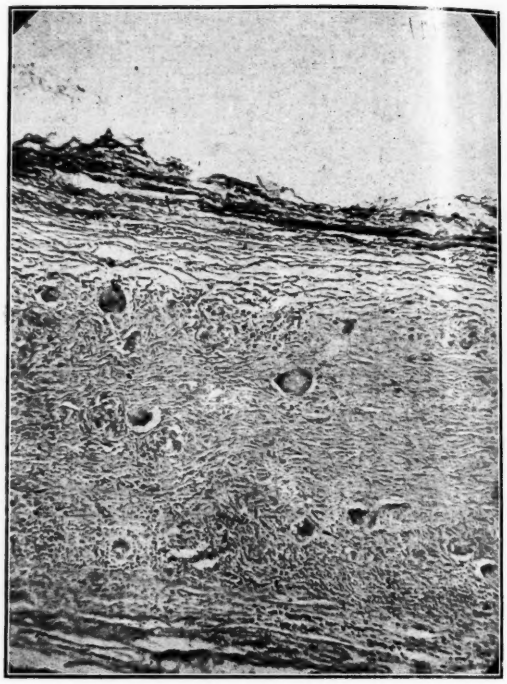

FIGURE VI.
Case II., H.C.P., Tuberculoid Leprosy. Subcutaneous Nerve of Leg. Cellular Infiltration and Giant Cells. × 65. Ziehl Neelson.

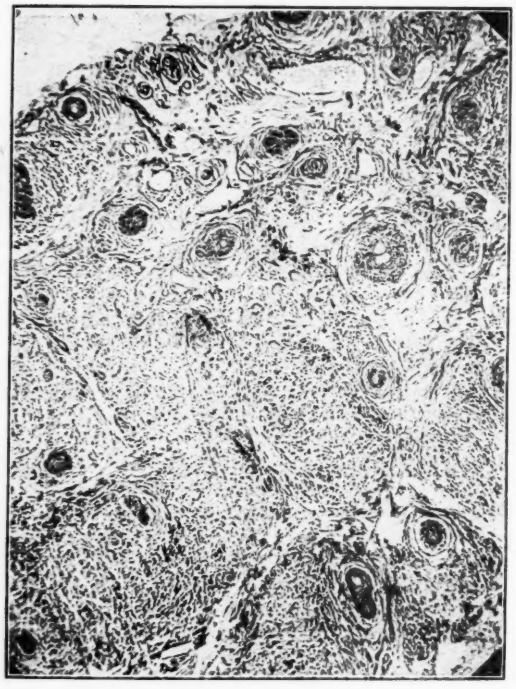

FIGURE VII.
Case III., Mrs. S., Doubtful Granulomatous Lesion. Sections of Skin of Face. Numerous hair follicles are cut obliquely. × 65. Hæmatoxylin and Eosin.

FIGURE VIII.
Case III., Mrs. S., Showing Three Giant Cells in the Infiltration. × 500. Hæmatoxylin and Eosin.

ILLUSTRATIONS TO THE ARTICLE BY DR. A. H. TEBBUTT.

FIGURE IX.
Case IV., C.T., Early Leprosy. Skin of Elbow. No Typical Langhans's Giant Cells in the Infiltration. × 65. Hæmatoxylin and Eosin.

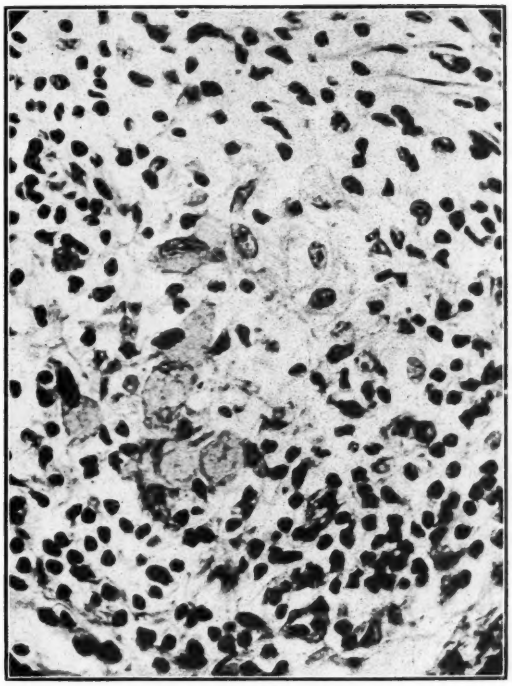

FIGURE X.
Case IV., C.T., Showing Epithelioid Cells and Small Round Cells. × 500. Hæmatoxylin and Eosin.

FIGURE XI.
Typical Leproma, Showing Enormous Numbers of Bacilli. × 250. Ziehl Neelson.

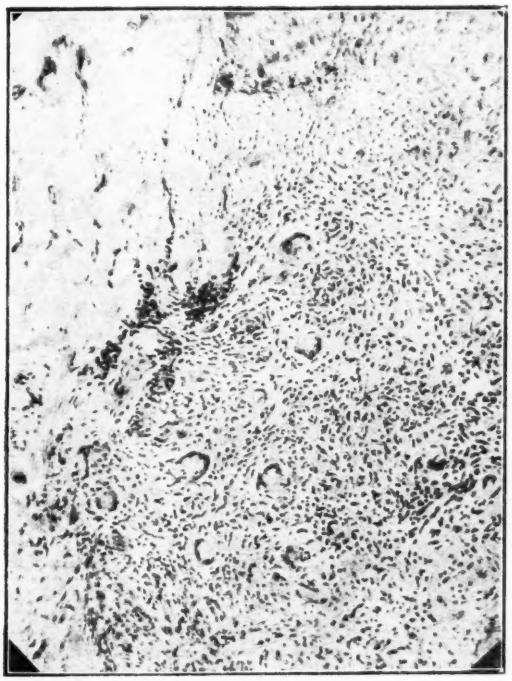

FIGURE XII.
Scrofuloderma Showing Giant Cells in the Infiltration. × Hæmatoxylin and Eosin.

ILLUSTRATIONS TO THE ARTICLE BY DR. E. H. STOKES.

FIGURE II.

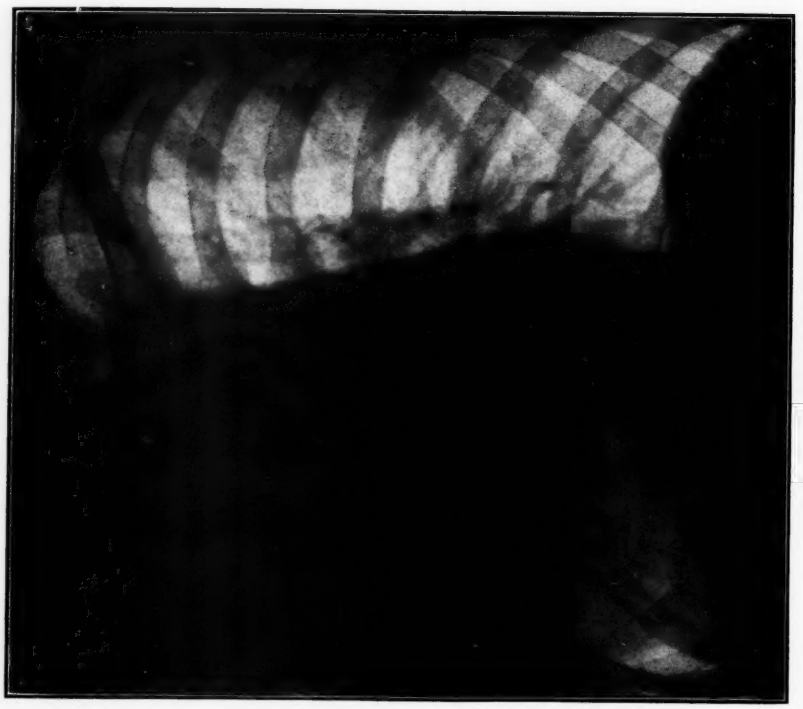

FIGURE I.

Appendix C.

Extracts Showing the Low Infectivity of Leprosy in the Case of Whites in the Southern Part of Australia.

Although lepers were imported to Victoria steadily during a long term of years and in considerable number, and although they always remained entirely unrestricted in their movements among the whites, no Victorian native white who had never left the colony, has ever been attacked. Moreover, the disease died away in Victoria quite independently of restrictive measures against the liberty of lepers, which in fact were first taken only in March, 1893.

Although coloured aliens of many different races, all of which have furnished cases of leprosy in Australia, have been imported during many years and in large numbers to all the better populated parts of the country . . . there is no evidence and no good reason for surmising, that any such native white has been attacked who lived south of the thirty-fifth parallel of south latitude.

I think, therefore, that a recent increase of leprosy on the continent of Australia has been alleged on superficial grounds, and that probably there has been no such increase.[17]

References.

(1) L. Rogers and E. Muir: "Leprosy," 1925.
(2) Virchow: "*Zur Geschichte des Aussatzes und der Spitälen*," *Archiv für Patholigsche Anatomie*, 1860, Volume XVIII., pages 138 and 273; Volume XIX., page 43; 1861, Volume XX., page 166.
(3) Newman: Proceedings of the New Sydenham Society, 1895.
(4) Cabanès: "*Mœurs intimes du Passé, Cinquième Série, Fléaux de l'Humanité*," page 200.
(5) Jonathan Hutchinson: "Notes on Leprosy in Various Countries," *The British Medical Journal*, March 22, 1890, page 651.
(6) "*Encyclopædia Britannica*," 1911, Volume XVI., page 479.
(7) Neuberger: "History of Medicine," page 39.
(8) E. Muir: "The Predisposing Causes of Leprosy," *The Lancet*, January 24, 1925, page 169.
(9) G. Archdall Reid: "The Laws of Heredity," 1910.
(10) Cecil Cook: "Leprosy in Australia," THE MEDICAL JOURNAL OF AUSTRALIA, September 27, 1924, page 336.
(11) E. Muir: "Leprosy, A Self-Healing Disease," *The Lancet*, February 9, 1924, page 277.
(12) Petrini de Galatz: "*Mittheilungen der Lepra-Conferenz zu Berlin*," 1897, page 438.
(13) Jadassohn: In Kolle and Wassermann's "*Handbuch der pathogenen Mikroorganismen*," 1913.
(14) "*Encyclopædia Medica*," Volume VIII.
(15) Macleod: "Diseases of the Skin," page 513.
(16) E. Muir: "Treatment of Leprosy," *Indian Journal of Medical Research*, October, 1923.
(17) Ashburton Thompson: "Prize Essay on Leprosy," New Sydenham Society, 1897.

TUBERCULOID LEPROSY.[1]

By A. H. TEBBUTT, D.S.O., V.D., B.A., M.B. (Sydney), D.P.H. (Oxon.),

Director of Pathological Department, Royal Prince Alfred Hospital, Camperdown, Sydney.

MY interest in leprosy and this slender contribution to its pathology are entirely due to the enthusiasm and persistence of Dr. E. H. Molesworth who enlisted my assistance in the histo-pathology and serology of some doubtful and to me difficult cases.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 7, 1926.

For those members of this Branch who have not had the pleasure of reading Leonard Rogers and Muir's very interesting and recent work[1] on leprosy I propose to abstract some of their main principles, then to pry into some other English and continental works with regard to tuberculoid leprosy and finally to give and illustrate the histo-pathology of the lesions seen by us.

Rogers and Muir classify leprosy into three types: (i.) Leprosy of the skin (skin leprosy), (ii.) leprosy of the nerves (nerve leprosy), according as the skin or the nerves are principally affected and (iii.) mixed leprosy in which both skin and nerve lesions are present.

They lay down three rules: (i.) The more the skin is affected, the less the nerves are involved and *vice versa*. (ii.) Nerve lesions are associated with a comparative paucity of the number of bacilli present in the body. (iii.) Consequently the earlier lesions of leprosy are chiefly of the nerve type, as there are few bacilli in the body at the outset and as the number of bacilli diminish towards the end of the disease, nerve lesions again tend to preponderate.

They also divide individual lesions into these three types. They regard the usual classification into nodular and maculo-anæsthetic types as confusing. A skin lesion is a skin area in which lepra bacilli can be found, but in which there is no anæsthesia to light touch. A nerve lesion is a skin area in which bacilli cannot be found, but in which there is anæsthesia to light touch. In one point of skin the finding of leprosy bacilli and anæsthesia to light touch are mutually exclusive, though one part of a lesion may show bacilli and another anæsthesia, constituting thereby a mixed lesion.

They describe three stages of leprosy: (i.) The first stage in which the bacilli are few; (ii.) the second stage in which the bacilli have multiplied and the disease is spreading; (iii.) the third stage is the stage of elimination or resolution. The bacilli break down and granulate and the lesions resolve.

The skin is infected either through an abrasion of the epidermis or by metastasis; that is to say, by a bacillary embolus in a skin vessel. When the bacilli at the point of infection are few in number, they are confined in their radial spread to the superficial layer. The degree of resistance is another factor which determines the variation in the nature and appearance of lesions. The more superficial the lesion, the more chronic it is; the fewer the number of bacilli present, the greater is the tendency towards nerve manifestations. They consider that a single lesion of the papillary layer of some skin area, not containing demonstrable acid-fast bacilli and associated with the ascending type of nerve lesion, is the commonest kind of primary lesion, if infection has been by the skin. Most patients when their condition is first capable of being diagnosed, have not become infectious or a danger to their associates, the diagnosis being made upon purely clinical signs, namely the presence of anæs-

thesia, nerve thickening *et cetera*, but bacteriological examination revealing no bacilli. The presence or absence of superficial sensation is thus the criterion of whether a lesion is to be classified as a nerve or a skin case. Any other line of demarcation between the two types of leprosy or lesions leads to countless difficulties.

The authors do not discuss the histology at length. Where the skin is infected by a radial spread through the papillary layer from the point of infection, there is flattening of the papillæ, the epidermal pegs largely disappear and there is a layer of fibrous tissue containing few bacilli. Beneath this is the infiltrated area containing round cells, plasma cells, connective tissue cells, dilated capillaries and bacilli in bundles and in twos and threes.

Where the skin is infected by a capillary embolus, the endothelium of the capillary is invaded and the endothelial and connective tissue cells proliferate. Radial invasions of the corium and a spread to deeper levels may follow.

The nerve lesions may be ascending or metastatic. In both there is proliferation of the connective tissue cells of the epi-, peri- and endoneurium and the formation of fibrous tissue, leading to compression of nerve fibres.

Why the bacilli enter the nerve sheaths more when there is a minimum of lepra bacilli in the skin and why they fail to enter at least to such an extent when there are far larger numbers of bacilli in the skin, it is difficult to say; but the fact remains that when smears taken from the corium reveal lepra bacilli, there is seldom superficial anæsthesia and when there is superficial anæsthesia, bacteriological examination of that part of the lesion which is anæsthetic, will reveal no bacilli.

Rogers and Muir recommend the use of "clip smears" in diagnosis. With a curved scissors a deep snipping is taken and smears are made from the deep surface. These are stained with hot carbol fuchsin for ten to thirty minutes, decolorized with 10% sulphuric acid or hydrochloric acid in alcohol for one to two minutes, washed in water and counter-stained with methylene blue. When granulation and gonidial forms are plentiful as compared with vegetative forms, the bacilli are being destroyed and the prognosis is improved.

It is useless, they contend, to examine an anæsthetic area, but if it have a raised erythematous margin which is not anæsthetic, a few bacilli may be found. Careful inspection of the skin and repeated "clip smears" may be necessary before the bacteriological test can be said to yield no result.

The question is raised whether there is not a non-acid-fast mycelial form of *Bacillus lepræ* which has not yet been distinguished histologically, but which is the main cause of nerve lesions and such mild radially spreading lesions as fail to manifest acid-fast bacilli on microscopic examination.

Smears from the nose are said to be of no great use. It is common to find bacilli in a clip smear and none in a nasal smear.

It is interesting to note that when bacilli are not found, a diagnosis based on the histological characters of a lesion in conjunction with the clinical findings is not discussed, nor is there any reference to giant cells or to tuberculoid leprosy.

Other English textbooks usually divide leprosy into two forms, nodular and anæsthetic and fail to refer to tuberculoid leprosy and even deny the presence of giant cells in uncomplicated leprous lesions. For example we find in Robert Muir's "Text Book of Pathology":[2]

> Some of the cells may contain a convoluted nucleus or multiple nuclei, but the giant cells, such as are common in tuberculosis, do not occur. Also there is no focal arrangement, no formation of follicles as in tuberculosis.

The same teaching is given in Muir and Ritchie's "Bacteriology."

In Manson's "Tropical Diseases"[3] it is stated that:

> The specific lesion of leprosy differs from that of tubercle inasmuch as the former is well supplied with blood vessels, contains no true giant cells and never undergoes caseation.

In Castellani and Chamber's[4] first edition we read:

> In the leproma sometimes typical giant cells, called Langhans's giant cells, are seen but these are really tubercular in origin.

But in the second edition this is corrected to the brief statement that:

> Sometimes typical giant cells, called "Langhans's" cells, are seen.

Macleod[5] writes in his "Diseases of the Skin":

> Certain of the lepra cells are enlarged and contain several nuclei, forming a type of giant cells which is different from the Langhans's giant cell of tuberculosis in that it has neither the peripheral arrangement of nuclei nor the necrotic centre. Giant cells simulating the Langhans's cells have been described from time to time in leprosy, but before accepting them, it is necessary to be certain that a mixed infection with tuberculosis did not exist.

Phineas Abraham, writing in Allbutt and Rolleston's "System of Medicine," gives a thorough account of the morbid anatomy:[6]

> Occasionally very large multinucleated cells, somewhat resembling the "giant cells" of lupus, are seen in the peripheral or deeper parts of the growth. . . . Hansen and Thin believe that when true "giant cells" are seen, the growth is tuberculous and not leprous. Thomas, Benoir and Boinet and others have also described large multinuclear cells as sometimes present in the leprous neoplasm. Bergengrün and Dohi's observations place the matter beyond doubt, *viz.*, that they do exist in lepromas. . . . . Sections taken by me from one of the erythematous macules in an early stage of the disease, in a boy at the Westminister Hospital, showed an infiltration of the corium with an irregular cell growth, in which some large multinucleated cells were also scattered. In this case no bacilli were then observed, although they were found in abundance in subsequently developed nodules.

Abraham does not allude to giant cell formation in lymph glands or nerves.

It may be that in English periodicals I have missed descriptions of tuberculoid lesions. In New

South Wales, however, as long ago as 1895, F. Tidswell[7] described a nerve lesion of this type in a case of mixed leprosy. Acid-fast bacilli were found in the eyebrow during life and *post mortem* the thickened peroneal nerve showed infiltration by:

> Round, oval and irregular cells for the most part mononucleated, but in one or two places multinucleated large cells, containing many nuclei (eight to twelve) were seen. Normal nerve fibres were scanty, the majority being degenerated. . . . Bacilli are very numerous.

Tidswell, however, regarded it as a rare lesion, in fact he appears not to have met with such a lesion before.

Even as far back as 1886 we find the French histologists, Cornil and Ranvier,[8] allude to islet formation and leprous tubercles, meaning apparently what now we usually call follicles. Sometimes:

> Large multinucleated and actual giant cells are found accumulated in old tubercles; these cells seem to us to result from the multiplication of nuclei and hypertrophy of the cells of the sweat glands.
>
> In older tubercles, large multinucleated cells, which have been called leprous cells by Virchow, are found in the leprous islets of the derma. These large, oval, spherical or irregular cells contain oval nuclei and are sometimes as large as the giant cells of tuberculosis, with which they might be confounded. They also contain an enormous quantity of rods.

I quote Hansen and Looft[9] to show that they had not recognized tuberculoid leprosy in Norway in 1895:

> In organs affected with tubercle one always finds, as is well known, giant cells and caseous degeneration; in the many, we can truthfully say, thousands of preparations of leprous affections which we have had under the microscope, we have never seen either a typical giant cell with marginal nuclei or caseous degeneration. There are indeed multinuclear cells in the lepromata, but never giant cells like those of tubercle. . . . The distribution of the bacilli in the tuberculous and leprous tissue is usually so very different, the tubercle bacilli being usually arranged singly, the lepra bacilli always in large quantities, in masses and clumps, that a confusion of the two diseases anatomically can only be possible in exceptional cases.

It is in German works that we find tuberculoid leprosy amply discussed and though the lesion is obviously a distinctive one, yet it is clear that it may represent only a phase in the evolution of either a nodular or maculo-anæsthetic case. The histological features should, however, be appreciated in English works; otherwise it is possible that the diagnosis of leprosy and thereby treatment may be either missed or unduly deferred.

Of authors using the German language one could not do wrong in putting Jadassohn first, for he writes on leprosy for Kolle and Wassermann.[10] He considers that the tuberculoid type of lesion in leprosy, with giant cells *et cetera* is proven and if it occurs in the skin and nerves, why should it not be found also in internal organs. Lesions in the internal organs should not be regarded as tuberculous superimposed upon leprous without good reason. Animal inoculation may be helpful.

In both tuberculosis and leprosy we find:

(i.) If bacilli are numerous, a non-specific inflammatory reaction, that is entirely or relatively devoid of giant and epithelioid cells. Examples of this are acute miliary tuberculosis, ulcerative tuberculosis of the skin and mucous membranes, nodular leprosy (the leproma).

(ii.) If bacilli are few, a typical tuberculous tissue reaction, for example subacute or chronic tuberculosis, tuberculoid leprosy.

(iii.) If bacilli are almost absent or not to be found, a non-specific inflammatory reaction, for example many of the so-called tuberculides, in some tuberculous joint infections, in *tuberculose sans follicule* of the internal organs and in the typical maculo-anæsthetic leprosy.

Type II. is common in tuberculosis, Types I. and III. are comparatively rare.

In leprosy Types I. and III. are well studied and common, whilst Type II. is (? hitherto) rare and atypical (possibly unrecognized or confused with tuberculosis and as Dr. Molesworth suggests, a phase in the development of racial and individual immunity). There occur in both diseases undoubted transitional and mixed forms.

I will pass by the elder Unna's work on leprosy[11] only alluding to one of his conceptions and that is this: that lepromata are developed in lepers whose whole cutis is a good culture medium for *Bacillus lepræ,* the nervous form in lepers whose subcutaneous nerves afford a suitable soil, but not the other tissues of the cutis.

There is an interesting discussion of tuberculoid leprosy by Paul Unna, junior,[12] in a recent publication. It is apparently by no means common in the big clinics in Germany for apparently only six cases had been seen in Unna's clinic since before 1912. In the opinion of Unna histological differentiation from tuberculosis may be impossible. Histological advances in the last ten years have broken down the arbitrary boundaries between the typical pictures of the three infective granulomata, syphilis, tuberculosis and leprosy. Experience has shown that in the differential diagnosis between these three the histo-pathology may in difficult cases fail to decide. Tuberculoid tissue reaction may occur not only in leprous and other forms of skin tuberculosis, but in a whole series of tuberculides, in leprosy, in secondary and tertiary syphilis, particularly in lichenoid syphilis, in sporotrichosis, oidiomycosis and even in actinomycosis. Of less typical form is the reaction around foreign bodies or about degenerate hair follicles. There is no cell element special to tuberculosis.

Jadassohn described tuberculoid leprosy in connexion with the maculo-anæsthetic form, but P. Unna, junior, finds that most examples including his own rather belong to the nodular type. One could, however, describe the lesions under consideration as constituting a third form of leprosy, the tuberculoid or lupoid variety, which though histologically nodular, has something in common with the maculo-anæsthetic form, namely the great scarcity of bacilli.

Tuberculosis and tuberculoid leprosy are by no means always to be differentiated histologically.

Unna agrees with Jadassohn that numbers of bacilli and amount of tissue reaction are in inverse ratio, the greater the primary reaction, the fewer the bacilli and the fewer the bacilli, the more chronic the tissue reaction.

Unna discusses also the results of inoculation experiments in monkeys. Quite frequently the tuberculoid tissue reaction has been found both in the internal organs and in skin papules. Doubtless, too, in many human autopsies in the past the finding of tuberculoid tissue in internal organs has caused an undue frequency of the diagnosis of a superimposed infection with tuberculosis.

Finally with regard to the development of the tuberculoid skin lesion, Unna does not agree with a suggestion of J. Kyrle, based on some monkey inoculations, that this histological picture may be the end result of a leproma. Rather would he agree with Lewandowsky and Jadassohn that destruction of bacilli and advent of antibodies produce tuberculoid tissue; the bacilli are few in number and they may have diminished virulence. Yet the tuberculoid lesion may through the multiplication of the bacilli progress later to a typical nodular lesion. This was shown by Kedrowski,[13] who described a maculo-anæsthetic case with so definite a tuberculoid histology that the diagnosis was for a time doubtful. After seven years the lesions were quite changed to the nodular type with numerous bacilli. Such may have happened by the way in our first case, Miss R.

Before proceeding to the description of our cases, it is at this point interesting to recall the contribution by Petrini de Galatz[14] in 1897 in which he discusses the survival of leprosy in Brittany. After describing several cases of leprosy in which bacilli could not be found, he quotes "our distinguished leprologist," Dr. Zambaco-Pacha, in his communication to the Academy of Medicine of Paris:

> In Brittany leprosy maintains occasionally all its classical characters, but in the vast majority of cases it is mild and attenuated and reveals itself by only one or two of its characters. In a word it is a *forme fruste* and in consequence unrecognizable by those not experienced enough to unmask it. It is a fact, too, that anæsthetic leprosy is arrested in the first stage for years and even altogether. In some cases bacilli cannot be found and it is necessary to make a clinical diagnosis. Many conditions said to be syringo-myelia and Morvan's disease in Brittany were really leprosy.

The Histo-Pathology of the Sydney Cases.

I will now deal with the histo-pathology in four selected patients. On the evidence brought forward I feel justified in classifying Cases I. and II. as examples of indigenous tuberculoid leprosy. Case III. (the patient being a native of Madras) presents a granulomatous lesion, possibly leprous, but unproven and is a good example of the difficulties of diagnosis. Case IV. presents early leprous lesions in which, though there is a convenient depth of infiltration, yet the bacilli were found only in very small numbers in sections and not at all by puncture.

Case I.: Tuberculoid Leprosy.

Miss R. was twenty years of age when first seen in 1917. Sections were cut from a large deeply pigmented infiltrated lesion on trunk. No reaction was obtained to the Wassermann test. She was not improved by antisyphilitic treatment. No bacilli were found in sections. A provisional diagnosis of tuberculosis was made. Four years later *Bacillus lepræ* was found and the patient was interned in the lazaret.

Histo-Pathology of Original Lesion.

The epidermis of the skin of the trunk is slightly thinned but the epidermal downgrowths or pegs are present and there is a ribbon-like infiltration in a wide area of skin, commencing immediately below the basal cell layer and extending down in the corium to about twice the depth of the average peg. The dermis beneath is a thick layer of normal fibrous tissue in which, however, are at intervals small granulomatous nodules. Therefore the papillary layer of the skin and a layer immediately beneath it contain the main lesion (see Figure I.). The infiltration here at first glance is diffuse, but there is a definite tendency to a follicular arrangement in which pale endothelial cells form the centre and frequently on one side of them are multinucleated giant cells and the periphery is formed by small round cells. Plasma cells, if present, are few in number. There are few hair follicles, but the infiltration may be seen around them and the deeper nodules are usually around atrophic sweat glands or vessels and sometimes a small cutaneous nerve is involved, its sheath showing several layers of connective tissue fibrils and endothelial and small round cells may be seen in its substance. The giant cells are most striking in the papillary layer, but are common throughout and as many as five were found in one deep nodule. They vary in size, usually the nuclei are very numerous and peripheral, sometimes they are massed on one side, sometimes they form a circle and again they may be both central and peripheral. The nuclei are vesicular and oval or somewhat elongated. The central mass of the giant cells is hyaline. A few giant cells contain or adjoin peculiar double-contoured rounded or oval bodies (see Figure II) usually staining deeply with hæmatoxylin. A similar unstained body was found apparently lying free in one nodule. What appeared to be a broken segment of lanugo hair was found lying free in the tissues near one hair follicle. Hæmatoxylin stained fibres in places beneath the epidermis and also around deep nodules and passing from these out into the fibrous cutis were thought to be altered elastic fibres. There is no evidence of necrosis or suppuration. No typical lepra cells were found nor were acid-fast bacilli found by the Ziehl-Neelsen method. I suggest that the peculiar double-contoured bodies are the remains of lanugo hairs. P. Unna, junior, has interpreted as such similar bodies in tuberculoid leprosy.

Case II.: Tuberculoid Leprosy.

H.C.P., male, was thirty to forty years of age. Sections were cut from two infiltrated (nodular) skin lesions, one a progressive lesion on the chin, also from a swollen submental lymph gland. No bacilli were found in these sections, nor in expressed serum or nasal smears. The serum did not react to the Wassermann test. Subcutaneous tuberculin and von Pirquet tests yielded no reaction. Guinea pig inoculations with tissue yielded no result. On the advice of Dr. Harold Dew a thickened nerve from the calf was excised and longitudinal sections manifested acid-fast bacilli. The patient was interned in the lazaret.

Histo-Pathology.

The epidermis of the skin of the chin is irregularly thinned and the epidermal pegs are blunt and widely separated. Commencing immediately beneath the epiderm and extending down into the subcutaneous fat is granulomatous tissue, incompletely lobulated by thin fibrous tissue bands and pierced by a few hair follicles (see Figure III.). Deeper still in the fat are a few nodules not showing any connexions with the main mass. Buried

in the granuloma are a few sebaceous glands with shrunken cells and also sweat glands, some of which are compressed. Examinations under low powers reveal numerous giant cells many of which are exactly similar to those in the previous case (Miss R.), but there are many also of smaller size, rounded, triangular or quadrilateral in section and with fewer nuclei. Several of the curious bodies previously described were also found (see Figure IV.), one being adjacent to a hair follicle and one is elongated showing that it is cylindrical and not spherical.

There is a more distinct follicular formation in the deeper parts than superficially. The predominant cells in the follicles are epithelioid cells with vesicular oval nuclei and much cytoplasm, sometimes showing a fine vacuolation, the vacuoles being smaller than those usually seen in the so-called lepra cells. Mature and immature giant cells often lie with these epithelioid cells and peripherally is not uncommonly a narrow ring or collection of small round cells. The deeper follicles also sometimes show a small vessel or some sweat glands or a nerve, not necessarily centrally placed. The nerves seen show definite fibrous replacement and the perineurium is thickened. A medium sized artery and vein show no infiltration of their walls. There is some fibrous tissue formation in the granuloma and hæmatoxylin-stained fibres irregularly distributed are considered to be altered elastic fibres. Plasma cells are comparatively few. There is no necrosis or suppuration. No acid-fast bacilli were found.

The submental lymph gland shown in Figure V. is obviously enlarged. The capsule is not thickened. The lymphoid structure is obscured probably by the general swelling. There is no thickening of the trabeculæ. So-called germ centres are not seen. Scattered through the whole section are small groups of epithelioid cells resembling those in the skin and forming small rounded nodules. In some of these are typical giant cells often with peripheral nuclei and occasional giant cells appear to be isolated amongst the small round cells. The nodules simply lie in the lymphoid tissue not surrounded by fibrous tissue, nor is there any vascular engorgement or other leucocytic infiltration. No acid-fast bacilli were found.

A thickened subcutaneous nerve in the calf of the leg, about 2·5 centimetres (one inch) long was examined (see Figure VI.). Some anæsthesia was present in the leg below the site of resection. Longitudinal section of the nerve shows some slight thickening of the sheath, not dense fibrous tissue, but loose bundles of connective tissue fibrils. The main thickening, however, is due to the cellular and connective tissue increase in the endoneurium. Though a few dilated *vasa nervorum* are to be seen, the nerve is relatively avascular, but there are numerous interstitial channels which may be lymph channels. Where a branch of this nerve is given off, the infiltration can be seen passing into it and the perineurium here is markedly infiltrated with connective tissue cells, mainly small round cells and giant cells, to form a thick collar.

A striking feature of the whole nerve segment is the number of giant cells. These resemble those in the skin and lymph gland already described. There are a few in the sheath, but they are more numerous in the substance of the nerve. Small round cells are not uncommon under the outer sheath. There are fibroblasts and an increase of connective tissue fibrils, but larger cells are very numerous both diffusely distributed and also aggregated into groups or into a follicular arrangement with or without giant cells, but not surrounded by small round cells. Some of these large cells, usually mononuclear and with varying amounts of cytoplasm, are doubtless plasma cells. Many others are endothelial cells and others are epithelioid cells similar to those in the other lesions, some showing a finely vacuolated cytoplasm (lepra cells). The cellular and connective tissue encroachment on the nerve fibres must be very considerable. A few nerve fibres could be seen, but no staining was employed to show the myelin sheaths.

Ziehl-Neelsen's method discloses a moderate number of acid-fast bacilli. These are not common in the sheath, but are found with ease in the endoneurium. They appear to be both intra- and extracellular, the latter mainly in relation to endothelial cells. They are very fine rods, often granular, or minute dots, possibly bacilli cut transversely. There are also larger rounded or irregularly shaped dots sometimes intracellular in a group and suggesting phagocytosis and degeneration.

Case III.: Diagnosis Doubtful.

The patient, Mrs. S., was thirty-six years of age. Sections were cut from the cheek, part of a somewhat symmetrical lesion on the face and forehead, showing soft infiltration at the edges and no ulceration. The serum had not reacted to the Wassermann test. Antisyphilitic treatment produced a doubtful slight improvement. Subcutaneous tuberculin tests yielded no reaction. No acid-fast bacilli were found.

Histo-Pathology.

The section from the skin of the cheek is cut obliquely through the skin. The epidermal pegs are not well seen and the epiderm is not thickened. Immediately beneath is a thin layer of fibrous tissue continuous with bands descending to surround hair follicles and granulomatous nodules (see Figure VII). This tissue extends deeply even into the subcutaneous fat and a follicular arrangement is more definite in the deeper parts. Superficially it is rather diffuse, but even here the central position of ordinary endothelial cells, cells with more cytoplasm, some of epithelioid type, similar cells with two nuclei and immature giant cells with three or four nuclei, are well seen (see Figure VIII.). The small round cells tend to be peripherally arranged. Plasma cells are few in number. Occasional giant cells were found with many nuclei, but they had not such a hyaline centre as in the two previous cases. Superficially some of the hair follicles are surrounded by a cellular infiltration. Dilated vessels with only an endothelial coat reach almost to the epidermis. They are probably blood vessels. The deeper nodules showing a follicular arrangement were most commonly found around small vessels, but sometimes about sweat glands which were somewhat atrophic, and sometimes about a small nerve. The latter showed definite fibrous tissue increase and a few cells of the infiltration could be seen in their interior. Fibroblasts were to be seen throughout the granuloma and fibrotic changes were probably active. In the subcutaneous fat small subcutaneous nerves showed fibrotic changes. The curious quasi-foreign bodies were not found in this case.

Case IV.: Early Leprosy.

C.T., a male, was fifty-six years of age. The first lesion appeared six months ago, the infection apparently originating in an accidental wound of one hand nine years before whilst employed in a lazaret. Lesions show pulpy infiltration. Sections were cut from a lesion on one elbow. The serum did not react to the Wassermann test. No acid-fast bacilli were found in repeated punctures, but a very few bacilli and coccoid forms were found in sections.

Histo-Pathology.

A section of skin from the elbow shows an epidermis with well marked *stratum corneum*, but with unthickened *rete mucosum* and the epidermal pegs and papillæ are few. There are not many hair follicles. Numerous cellular foci are scattered through the dermis and these or prolongations of them are seen immediately under the epidermis (see Figure IX). The intervening connective tissue is rather dense and fibrous. The follicular arrangement of the granulomatous tissue is a striking feature. The superficial follicles tend to show more small round cells than the deeper, but almost all manifest in varying numbers endothelial cells in a central position. Some of the endothelial cells have a large amount of cytoplasm with small or large vacuoles. The nucleus is usually vesicular. Some would be described as epithelioid cells and resemble the large vacuolated vessels of a leproma (see Figure X.). The follicles are formed mainly around small vessels which are often situated towards the periphery of the follicle. Some of the deeper follicles are around sweat glands. Indubitable nerves were difficult to

find, but in one of the largest follicles a small nerve cut transversely showed increase of fibrous tissue and some connective tissue cells in its cross section.

None of the curious quasi-foreign bodies previously described were found. No typical giant cells were noted, but some endothelial cells contained two or three or even four nuclei. In one section completely searched I found one small clump of acid-fast bacilli, apparently intracellular, in a deep follicle. In several cells the cytoplasm showed numerous minute acid-fast granules which may be disintegrated bacilli. There were also mainly in the cells of sweat glands small acid-fast spore-like bodies, varying in size up to that of a staphylococcus. It is possible that these latter are artefacts, but such granules have been described in sweat glands in leprosy. Unna considered that the bacilli are disintegrated in sebaceous and sweat glands and never reach the surface by these routes.

The Wassermann Reaction in Leprosy.

It will have been noted that these four patients gave no reaction to the Wassermann test and this negative evidence was undoubtedly of considerable diagnostic value, for Dr. Molesworth says "the first impression is that usually experienced on inspection of a syphilitic lesion." Yet it is an irritating and not uncommon experience to hear some clinicians decrying the value of an indubitable absence of reaction in a case for diagnosis.

Dr. Badham informs me that some few years ago most of the lazaret patients in Sydney failed to give any reaction, but the antigen in use at that time was not particularly sensitive.

Dr. P. Fiaschi has reminded me of another puzzling case of a patient whose lesions were suspected to be syphilitic and whose serum was found to give a strong Wassermann reaction, but who failed to respond to intensive antisyphilitic treatment. He was compelled to reconsider his diagnosis and an excised lesion disclosed numerous acid-fast bacilli. Treatment as leprosy produced considerable improvement. I am not in a position at present to comment further on this case, pending further investigations. Impey had much to say in the pre-Wassermann days on the association of leprosy and syphilis; in fact he makes a fourth type called "syphilitic leprosy," apparently a grave condition at that time in South Africa. Perusal of leprosy statistics shows a large percentage of Wassermann reactions, but Kolmer in his papers, quoted by Rogers and Muir, claims that using an antigen free from anticomplementary activity, a reaction is never obtained unless leprosy is accompanied by syphilis. He also states that the Kahn test will give almost as distinctive results. His figures are worth reproducing.

Patients.	Wassermann Test.			Kahn Test.		
	+		−	+		−
23 lepers without evidence of syphilis	0	..	23	2	..	21
2 lepers with lesions suspected to be syphilis	2	..	0	1	..	1
3 lepers definitely syphilitic	3	..	0	3	..	0

Other investigators have recently confirmed this view. It appears likely therefore that with acetone-soluble-free extracts the Wassermann reaction is usually absent in leprosy and if present, syphilis should be strongly suspected.

I take this opportunity of thanking Dr. Molesworth and Dr. Harold Dew for indispensable advice; Drs. Millard and Morgan for material; Dr. V. Woodhill, Messrs. Dawes and Burfield for patient assistance; Drs. E. W. Ferguson and F. Tidswell for books and reports.

Summary and Conclusions.

1. Some aspects of Rogers and Muir's teaching on the pathology and diagnosis of leprosy have been summarized for Australian readers.

2. The omission and even denial of tuberculoid leprosy in English standard works should be corrected in the light of German work of many years' standing. The recognition of this type of lesion will tend to prevent mistakes in early diagnosis in some difficult cases. The histological method should be employed, as well as a bacteriological search and the Wassermann test should be carried out. I recognize also from our experiences that a careful clinical examination is all important, the finding of anæsthesia in particular.

3. The lesions of tuberculoid leprosy in Australian white people are described. A doubtful case and an early nodular case are also included.

References.

(1) L. Rogers and E. Muir: "Leprosy."
(2) R. Muir: "Text Book of Pathology," 1924.
(3) Manson-Bahr: "Manson's Tropical Diseases," 1921.
(4) Castellani and Chambers: "Manual of Tropical Medicine," First Edition, 1910; Second Edition, 1913.
(5) Macleod: "Diseases of the Skin," 1920.
(6) Phineas Abraham in Allbutt and Rolleston's "System of Medicine," 1907, Volume II., Part II., page 662.
(7) F. Tidswell: "Government Report on Leprosy in New South Wales for the Year 1895," page 15.
(8) Cornil and Ranvier: "Pathological Histology" (English Edition), Volume II., Part II., 1886.
(9) Hansen and Looft: "Leprosy" (English Edition), 1895.
(10) Jadassohn in Kolle and Wassermann's *"Handbuch der Pathogenen Mikroorganismen," Band* V., 1913.
(11) P. G. Unna: "Histo-Pathology of the Diseases of the Skin" (English Edition), 1896.
(12) P. Unna: *"Beitrag zur Frage der Tuberculoiden Lepra," Virchow's Archiv für Pathologische Anatomie und Physiologie und für Klinische Medicin, Band* 246, 1923.
(13) W. Kedrowski: *"Zur Histologie der Lepra," Archiv für Dermatologie und Syphilis,* 123, *Seite* 1.
(14) Petrini de Galatz: *"Mittheilungen der Lepra-Conferenz zu Berlin,"* 1897.

Reports of Cases.

HYDATID OF THE LUNG.

By E. H. Stokes, M.B., Ch.M. (Sydney),
Honorary Assistant Physician, Sydney Hospital, Sydney;
With a Note on the Serological Findings,
By C. H. Shearman, M.B., Ch.M. (Sydney),
Serologist, Sydney Hospital, Sydney.

Clinical History.

H.F., a single man, aged twenty-three years, by occupation a driver, was first seen at the medical out-patient department of the Sydney Hospital on May 7, 1925, and

was referred to the Medical Superintendent for admission with the provisional diagnosis of pulmonary hydatid.

He was admitted on May 14, his chief complaint being that he was suffering from weakness which had increased during the previous four months. He stated that nine months ago he suffered from an attack of pleurisy (diagnosed as such by a medical man) which arose after he received a blow on the left side of the chest from a falling case of bananas. After recovery he returned to work and was well till four months previous to admission. During those four months he had lost 6·3 kilograms (one stone) in weight, had suffered from slight cough and had felt tired in the afternoons. His appetite, however, had remained good; he had slept well and had been able to do his ordinary work up till the day of admission. The amount of sputum was small; it was sometimes white, sometimes yellow and often blood-stained during the last six months, the last occasion being six weeks prior to admission. He did not suffer from any pain and he became breathless only when performing heavy work; he had no rigors and no night sweats.

There was no evidence of tuberculous infection in his family. He was afflicted with infantile paralysis at eighteen months of age; this left him with wasting of the right leg. No history of venereal disease was elicited. He stated that he had worked on a sheep station in New Zealand some years previously.

On physical examination it was noticed that although pale in appearance, he was well nourished; his temperature was 37·2° C. (99° F.), the pulse rate was 100 and the respiration rate 30 per minute. There was a very darkly pigmented mole about 2·5 centimetres (one inch) in diameter on the back of his neck. On inspection of his chest it was found that the respiratory movement on the left side was restricted. All the left side of the chest (with the exception of the lowest portion of Traube's space) anterior to the posterior axillary line was dull to percussion. Over this dull area the tactile fremitus, the breath sounds and the vocal resonance were absent, but posterior to the posterior axillary line the left lung was normal except at the apex, where there was an area of comparative dulness with faint vesicular breathing.

The cardiac impulse was displaced to the right, being in the third and fourth right interspaces 7·5 centimetres (three inches) from the midline.

The chest was examined by X rays and Dr. W. A. Edwards reported the presence of either hydatid or new growth of the lung (see Figure I.).

The various pathological examinations for hydatid disease were performed by Dr. Shearman. The intradermal test of Casoni elicited no response, either immediate or delayed, nor did the blood serum give any reaction with either the complement deviation test (hydatid antigen) or the precipitin test.

The blood count on May 14 yielded the following result:

Total number of red blood cells	5,800,000
Hæmoglobin value	85%
Colour index	0·7
Total number of leucocytes	13,000
Polymorphonuclear cells	68%
Lymphocytes	28%
Eosinophile cells	0%
Basophile cells	0·5%
Large mononuclear and transitional cells	3·5%

His sputum was reported on as follows: "No tubercle bacilli were seen, but staphylococci, streptococci, pneumococci, *Micrococcus catarrhalis* and Gram-negative bacilli were present."

The Wassermann test was performed on May 14 and the following report was given by Dr. Shearman: "(i.) Ice box method: The serum is somewhat anticomplementary, but the indications point to a reaction being present. (ii.) Warm method: A fairly strong partial reaction to the test was present."

On May 27 the serum was again tested for reaction to the Wassermann test. It was still anticomplementary with the ice box method of technique, but a strong partial reaction was again elicited by the warm method.

On May 27, a fortnight after the first blood count, his leucocytes numbered 21,000 (polymorphonuclear cells, 61·6%; lymphocytes, 35·1%; eosinophile cells, 1%; basophile cells, 0·3%; large mononuclear and transitional cells, 2%).

His larynx was found by Dr. D. G. Carruthers to be normal.

The temperature chart showed evening rises to 38·3° C. (101° F.), the pulse rate varied between 80 and 100 per minute and the respiration rate between 20 and 40 per minute.

The case thus presented many anomalies and it was thought that the differential diagnosis lay between the three following conditions: Encysted pleural effusion, hydatid of the lung, malignant neoplasm arising either in the pleura (for example, endothelioma) or in the lung (for example, carcinoma) or in the mediastinum (for example, sarcoma).

(i.) In regard to encysted pleural effusion, the displacement of the heart and the physical signs were compatible with the presence of encysted fluid and the increase of the leucocytes from 13,000 to 21,000 suggested the presence of pus.

(ii.) In regard to hydatid of the lung, the negative tests for hydatid disease and the absence of eosinophilia were against this diagnosis, but nevertheless, the possibility of hydatid could not be excluded.

(iii.) In regard to malignant neoplasm, the absence of cachexia, the fact that there was no evidence of increased intrathoracic pressure (such as dilated superficial veins, pain and dyspnœa) and the appearance of blood stained sputum only occasionally were all against neoplasm. Although the marked displacement of the heart would also be unexpected in this condition, leucocytosis not uncommonly accompanies a new growth. The pigmented mole on the neck could hardly be regarded as a primary focus for a secondary infection of the intrathoracic contents.

In view of the Wassermann reaction syphilis had to be excluded, but one could hardly imagine syphilitic lesion of the lung reaching the size the physical signs indicated without ulcerating or caseating.

The most likely diagnosis appeared to be encysted pleural effusion and it was thought that the correct procedure would be for a surgeon to explore the chest while the patient was anæsthetized because of the possibility of the presence of hydatid of the lung.

On account of the unusual physical signs and the extreme difficulty of arriving at a diagnosis the patient was seen in consultation by Dr. J. M. Gill, Dr. H. J. Ritchie, Dr. A. W. Holmes à Court and Dr. L. W. Dunlop (Honorary Physicians), Dr. G. C. Willcocks, Dr. W. Evans, Dr. J. G. Hunter and Dr. R. C. Winn (Honorary Assistant Physicians) and by Dr. H. Hamilton Marshall (Honorary Consulting Physician) and afterwards shown at a meeting of the Sydney Hospital Clinical Society held on May 28, when it was decided that the chest should be explored by a surgeon with the patient anæsthetized.

On June 3, by which time the patient had lost a further 2·2 kilograms (five pounds) in weight, all doubts as to the diagnosis were set at rest, as he coughed up several ounces of purulent semi-solid sputum containing grape-skin-like bodies which on microscopical examination proved to be hydatid ectocyst. No hooklets and no scolices were found.

On June 8 a second skiagram was reported upon by Dr. P. S. Parkinson as follows: "Left side of chest radio opaque, dense shadow with lower rounded margin appearance very suggestive of large hydatid of the lung." The appearance is reproduced in Figure II.

On June 13 he coughed up more hydatid cyst wall and there was a further diminution of the area of dulness over Traube's space.

On June 19 he was operated upon by Dr. Archie Aspinall, ether being administered by the intratracheal method by the Medical Superintendent (Dr. Winston). An oblique incision was made extending from the second left rib in the parasternal line, to the seventh left rib in the mid-axillary line. The *pectoralis major* was split in the line of its fibres. The periosteum was cleared from the fourth and fifth ribs and about 7·5 centimetres (three inches) of

each excised. An attempt was made to stitch the parietal pleura to the underlying structures, but fluid like hydatid fluid escaped, so a large needle was entered and turbid reddish fluid withdrawn. The cyst was incised, the fluid evacuated and the cyst wall removed. The pleura was closed and a strip of iodoform gauze placed over the suture line. The muscles were sutured, the tissues superficial to the pleura being drained by means of a medium rubber tube. The skin was closed with silk worm gut and horse hair and the patient returned to the ward in good condition.

The gauze was removed next day and the drainage tube in a few days. Except for an hæmoptysis (about three hundred and sixty cubic centimetres) on July 6, the patient made an uninterrupted recovery and was discharged on July 31. Before discharge it was found that the apex beat was ten centimetres (four inches) to the left of the mid-line and that the breath sounds were present all over the left lung. He was examined by X rays on July 26 and the report by Dr. M. Frizell was that the heart shadow was normal. There was an area of thickened pleura extending from the left hilar region laterally to the outer aspect of the lung field. The elevation of the diaphragm was normal.

The blood serum now gave a reaction with both the complement deviation test (hydatid antigen) and the precipitin test, while the reaction to the Wassermann test was still present.

Comment.

The patient was no doubt infected with hydatid disease while employed on a sheep station in New Zealand some years previously. The fact that the cyst could grow to such an immense size and yet manifest so few symptoms was one of the most remarkable features of the case. It is difficult to decide whether the cyst arose primarily in the lung and later encroached on the pleural cavity or whether it was a cyst of the pleura. (Dr. Aspinall is inclined to the latter view.) The success of the operation was mainly due to the closure of the cavity after the removal of the cyst and the consequent prevention of infection. With regard to the reaction to the Wassermann test the patient denied the possibility of venereal disease and there was no clinical evidence of any past or present syphilitic disease.

Acknowledgments.

I wish to thank Dr. Archie Aspinall for the successful performance of the operation; Dr. W. A. Edwards for advice with regard to the skiagrams and also Dr. Bennett and Dr. King (Resident Medical Officers) for assistance with the notes of the case.

Note on the Serological Findings.

As frequently occurs in cases of pulmonary hydatid infestation, no reaction to the complement deviation test was elicited in this case reported by Dr. Stokes. This was not due to destruction or disappearance of antigen in the cyst fluid, as subsequent tests with the fluid proved it to have definite antigenic properties. Further, a few weeks after operation the patient's serum showed a reaction to the complement deviation test. Repeated tests made since the operation showed the reaction to increase in strength up to a period of four months after operation. On the last occasion of testing (January, 1926) the reaction was still manifest, though not so well marked as on the previous occasion.

These results are in accord with those noted by Mr. H. Dew and Miss F. Williams in recording their experience of the serological reactions in hydatid disease in Melbourne. Lack of absorption of the specific protein in the cyst fluid was undoubtedly the cause of the failure to react to the test before operation in this case of Dr. Stokes. The cyst when removed did not contain daughter cysts, nor was the adventitia thickened, indicating that there had been little or no trauma to the cyst before operation and therefore no leakage of specific protein. Within a few days of operation with its consequent letting loose of antigen the patient's serum began to yield a reaction.

The reaction of the patient's serum in the Wassermann test tended rather to confuse the issue. With the ice box method this serum absorbed by itself a certain amount of complement, that is, it was slightly anticomplementary, so that though in the presence of Wassermann antigen all the complement was destroyed, one could not give a definite opinion. When the serum was tested by the warm method of technique, a strong partial reaction occurred, suggesting a specific infection. Subsequent tests repeated on several occasions since operation have all shown a complete reaction to be present, so that it would appear that in addition to having had a hydatid infestation, a specific infection is also present, though probably in a latent state.

Reviews.

GOITRE.

THE Beaumont Foundation Lectures (Series 4), 1925, delivered under the auspices of the Wayne County Medical Society by C. H. Mayo and H. W. Plummer, of the Mayo Foundation, have been published in a small book.[1] One half of the book is written by Mayo and the other half by Plummer. The former commences by giving a brief survey of goitre and its recognition from the earliest time to the present day, followed by statistics of the number of patients treated at the Mayo Clinic suffering from goitre and the number of operations performed on them from 1892 to 1925. The anatomy and physiology of the thyreoid gland are briefly outlined. In tracing the development of the thyreoid gland the author states that the fœtal thyreoid is subject to adverse conditions and that it contains little or no iodine and that in new-born babes the gland contains iodine only if it has previously been administered to the mother. The infant obtains (in part) thyreoid secretion from its mother's milk. After consideration of the geographical distribution of goitre and its incidence according to race, age and sex, the remainder of the lecture deals with metabolism. From this the interesting fact emerges that when a patient suffering from exophthalmic goitre is placed upon a diet in which the protein is between thirty-four and one hundred and thirty-six grammes, it is possible to maintain a positive nitrogen balance.

The second half of the volume is from the pen of Plummer and it consists of three sections dealing with the classification of goitre, hyperthyreoid states and dysfunction of the gland respectively. Anatomically he classifies pure types under the headings of diffuse hypertrophy, diffuse colloid and adenomatous. The endemic goitres may be divided into diffuse colloid and adenomatous, the latter with and without hyperfunction. The lack of iodine and the alternate stimulation followed by colloid storage eventually results in hypertrophy (adenoma) with consequent hyperthyreoidism.

In exophthalmic goitre intensive stimulation of unknown origin of the whole gland, probably normal at first, causes the output of an excess of thyroxin and in addition an abnormal thyreoid product. This results in a diffuse hypertrophy and it may also alternate with a colloid storage stage. Considerable confusion of the histological picture is common, for the same glandular picture may be present at one time or another in either endemic or exophthalmic goitre. In the section on hyperthyreoid states the differences between exophthalmic goitre and adenomatous goitre are fully discussed and in the section on dysfunction Plummer states that he firmly believes that exophthalmic goitre is due to an excess of thyroxin and to an incompletely iodized thyroxin molecule. Plummer presents this two-product theory in an attractive light and describes the beneficial effects following the administration of iodine in exophthalmic goitre. The booklet is a *résumé* of the authors' experiences and opinions and is to be recommended to all interested in the subject.

[1] "The Beaumont Foundation Lectures—Subject: The Thyroid Gland," by Charles H. Mayo, M.D., and Henry W. Plummer, M.D.; Series Number Four; 1926. St. Louis: The C. V. Mosby Company. Post 8vo., pp. 83. Price: $1·75 net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 18, 1926.

Segregation for Infective Diseases.

If every person and animal suffering from a given infective process could be isolated from the remainder of the human and animal community and if a careful watch could be kept for the early manifestations of the same disease in those who appeared previously to be healthy, so that the isolation could be extended to harbourers of infection as well as to sufferers, it should be possible to stamp out that disease within a generation. This method of combating an infection has been attempted on many occasions, but has failed because it is impossible to fulfil the conditions set out above. In the instance of the acute infective processes epidemic spread can be checked to some extent by means of isolation. Experience has shown that the infection may be so slight that it is not recognizable and further the infecting agent may remain latent in some portion of an immune child's body. At a later date the infection may pass to a susceptible child and a fresh epidemic will result. It therefore follows that something more than isolation is needed to conquer even the acute zymotic diseases. Isolation by itself for the more chronic infections and particularly for those with long incubation periods is still less promising. The factors that militate against success are so many that in practice this measure is of little or no value. In the first place the diagnosis must be made before the patient has become a menace to his neighbours. Sometimes the diagnosis is difficult; sometimes the sufferer evades medical examination until the disease is far advanced; sometimes the medical practitioner neglects to employ all the means at his disposal to enable him to arrive at an early diagnosis. In the second place the named diseases are so common that it would be difficult to secure sufficient accommodation. Obstacles have been placed in the way of the notification of every tuberculous and syphilitic infection. The returns are notoriously incomplete, for it is believed by many that the information is not treated in a confidential way. Every man fears that he may become a pariah if it be known that he is suffering from tuberculosis or syphilis, that he may endure economic and social ostracism. But if to the expedient of notification were added that of isolation or segregation, the condition of every infected person would be regarded as intolerable. Acquired syphilis naturally carries with it a stigma, the mark of a breach of a social convention. The syphilitic will therefore do all he can to hide his infection. But there is no disgrace to be tuberculous. Nevertheless there is a desire on the part of the consumptive's relatives to conceal the fact of the infection. These reasons are sufficient to place beyond doubt that isolation and segregation cannot be applied as mass measures for the control of tuberculosis or syphilis.

Dr. E. H. Molesworth demands in his powerful article published in this week's issue that the endeavour to control leprosy by segregating all sufferers from this disease should be discontinued. The arguments used are unanswerable. If lepers were as common in Australia as consumptives, this measure would be refused on social, economical and humanitarian grounds. But since there are so few Europeans with this disease in our six States, segregation is tolerated. The handful of unfortunates is impotent. Notwithstanding the teaching of the thirteenth chapter of *Leviticus,* in which *Zara,ath* is described as the result of a social-ethical offence and in which the patient is condemned as unclean, notwithstanding the persistent exclusion of lepers from ordinary human intercourse from the earliest days of the recognition of the disease caused by Hansen's bacillus to the present time, a leprous infection is a misfortune, not a disgrace. What could be more cowardly than the persecution of these unfortunate people? Dr. Molesworth and many others have shown that segregation is useless to prevent the spread of this disease. But even if it served some purpose in the instance of persons with lesions discharging bacilli, it is evident that the majority of lepers do not disseminate the infec-

tion in a direct manner. Dr. G. H. Taylor showed many years ago that mild forms of leprosy are probably much commoner than was generally believed. Muir and Rogers maintain that leprosy is to some extent a self-healing disease. These are additional reasons against the adoption of segregation as a mass remedy. This expedient was conceived in error; it is the survival of a superstitious belief, devoid of all foundation in fact. Its continuation is a reproach to an enlightened community. Our lazarets should be immediately converted into refuges for patients in the terminal stages of the disease, places where they may find some release from their pain and distress. But they must no longer be prisons into which the patients are cast against their free will. The doors of our lazarets must remain open to receive those who wish to enter and to permit those who wish it, to depart. Segregation as a measure for the combating of a chronic disease has been tried and has been found wanting.

Current Comment.

SYMPATHETIC INNERVATION AND SKELETAL MUSCLE TONUS.

THE experimental work of Royle on the sympathetic innervation of striated muscle, published in this journal in January, 1924, served to focus the attention of investigators throughout the world on the sympathetic nervous system. He and Hunter upheld the view, originally expressed by De Boer and advocated by Langelaan, that plastic or postural muscle tonus is maintained by the sympathetic nerve fibres ending in voluntary muscle. When Royle and Hunter went to New York to make this work the subject of their Doctor John B. Murphy Oration, further prominence was given to it and many surgeons tried to obtain results identical with those described by Royle. It is not necessary to describe these in this place. Readers of THE MEDICAL JOURNAL OF AUSTRALIA are familiar with the claims made for sympathetic ramisection and many have seen patients who have benefited by its adoption. That certain surgeons have not produced satisfactory results in dealing with patients does not prove anything. It is more than likely that such surgeons make an injudicious selection of patients and it is not beyond the bounds of possibility that the necessary rami are not divided. The fact that one observer obtains a certain result after the section of sympathetic rami and obtains no result in several consecutive cases under apparently similar conditions, points in all probability to an error in technique. This, however, is not important at the present juncture. The important point is the truth or otherwise of the physiological principle that plastic tonus is maintained by the sympathetic fibres in voluntary muscle.

Royle carried out certain experiments on the goat and Hunter recorded observations after sympathectomy in fowls. In both sets of experiments loss of tone was manifest and on these experiments the conclusion in regard to the physiological principle was based. Important confirmation of these observations has been recorded recently by Dr. Albert Kuntz and Dr. Alver H. Kerper.[1] Not only have they carried out confirmatory experiments, but they have done what is much more valuable, they have recorded some of their observations graphically. Their first set of observations was carried out on upwards of twenty dogs. The sympathetic trunk on one side in the lumbar region was extirpated. In some animals it was found that a degree of hypotonus could be palpated in the muscles which were deprived of their sympathetic nerve supply. They found many animals in which this was not possible. In view of their more accurate observations to be described later, they did not regard this fact as of material importance. In no instance was any impairment of function or weakness of muscles to be detected in the normal behaviour of the dogs. When the animals were anæsthetized, flaccidity at once became apparent in the muscles supplied by the excised sympathetic nerves. In two animals no change was found. At *post mortem* examination it was found that the left sympathetic trunk had been successfully extirpated. Dr. Kuntz and Dr. Kerper can find no explanation for this. In regard to the other animals they suggest that the somatic innervation in large measure compensates for the loss of the sympathetic innervation in the maintenance of normal posture and that the somatic nervous mechanism is more profoundly affected by anæsthesia. They also regard it as probable that in this compensating effect of the somatic proprioceptive mechanism lies the explanation of many of the recorded failures to recognize any influence of the sympathetic innervation on skeletal muscle.

The second set of experiments was carried out on birds. These were mainly a repetition of Hunter's experiments (see THE MEDICAL JOURNAL OF AUSTRALIA, June 14, 1924) and are held by Dr. Kuntz and Dr. Kerper to corroborate Hunter's results.

The experiments of the third group were those concerned with actual measurement. Dr. Kuntz and Dr. Kerper are quite correct when they state that "additional data which approach quantitative accuracy, are urgently needed." The method employed by them is that used by Spiegel in 1923. The *quadriceps femoris* was used on account of its convenience for mechanical reasons and because it is an extensor muscle concerned in the maintenance of posture of a limb.

[1] *The American Journal of Physiology*, March 1, 1926.

The apparatus consists of an ingenious arrangement of rods, counter weights and hinges placed so that rotation of the hinge of the apparatus corresponds to the degree of flexion of the knee. The angle of rotation is read off on a protractor. Mathematical formulæ are given for the estimation of the tension of the *quadriceps femoris* muscle. A tonus curve is plotted by use of the different values of this tension as calculated for the successive position of the limb for the abscissæ and the angles of flexion for the ordinates. It was found that this tonus curve or the curve of passive extension of the quadriceps muscle of the normal animal rises very slowly at first and then more rapidly as the angle of flexion increases. This indicates that when the muscle is near its minimum normal length in the resting position, it offers greater resistance to postural change than after its length is increased by passive displacement of the leg. This phenomenon is common to muscles which normally maintain posture against the force of gravity. Rieger described it and applied to it the term *Bremsung* or brake phenomenon. Dr. Kuntz and Dr. Kerper point out that the brake phenomenon illustrates the capacity of the normally innervated muscle to resist extension while in a resting position independently of the nervous processes which initiate and control active contractions. It therefore involves the plastic tonus of Sherrington. They came to the conclusion that if plastic tonus were mediated through the sympathetic nervous system, the brake phenomenon might reasonably be expected to be absent in muscles deprived of their sympathetic innervation. A series of experiments was then carried out for the purpose of detecting a relationship of the brake phenomenon to the sympathetic innervation and to obtain comparative quantitative measurements of this type of muscle tonus before and after extirpation of the sympathetic nerve. More than twenty-five animals (cats and dogs) were used in this series. It was found that the brake phenomenon was absent in a quadriceps muscle which had been deprived of its sympathetic nerve supply, in other words the tonus of the muscle was materially reduced. In order to check the results still further nicotine was administered in some instances after the tonus of both quadriceps had been measured in animals which had been subjected to unilateral extirpation of the lumbar sympathetic trunk. In these circumstances the tonus curve was identical for each leg and coincided with the tonus curve produced before administration of the drug by the quadriceps deprived of its sympathetic nerve suppiy.

It is well known that the cerebellum plays an important part in the maintenance of postural tonus. In order to secure information on the relationship of cerebellar tonus to the sympathetic nervous system a series of experiments was carried out. The cerebellum was removed from animals in which unilateral extirpation of the sympathetic trunk had been performed and whose quadriceps tonus had been measured. It was found that the tonus of the quadriceps muscle suffered no further reduction. Similar findings attended destruction of the vestibular nuclei.

In discussing this work, Dr. Kuntz and Dr. Kerper point out that Royle and Hunter based their conception of plastic tonus on Sherrington's findings. They regarded it as a holding mechanism, as that component of tonus which enables a muscle to take up a new length without appreciable change in tension. Recent investigators have in their opinion assumed that the view advocated by Royle and Hunter implies two distinct kinds of muscle tonus, each of which is mediated through a distinct system of peripheral nerve fibres. Since decerebrate rigidity is a manifestation of exaggerated postural tonus, it has been argued that if plastic tonus is mediated through the sympathetic nerves, decerebrate rigidity ought to be entirely absent in a limb deprived of its sympathetic innervation. This has given rise to a tendency to discredit "the most important conclusions" of Royle and Hunter. Decerebrate rigidity is a tonic phenomenon whose plastic and contractile components have not been clearly differentiated. Moreover, Ranson's observation that decerebrate rigidity is reduced by the application of a solution of nicotine to the exposed dorsal root ganglia of the corresponding spinal nerves suggests that there is a nervous mechanism involved in the production of decerebrate rigidity which differs both from the ordinary somatic proprioceptive reflex arcs and from those which reach the muscles through the sympathetic rami. Dr. Kuntz and Dr. Kerper regard it as probable that decerebrate rigidity may not be well adapted to the study of the determination of the exact rôle of the sympathetic nerves in the production of muscle tonus. They go on to say that apparently the sympathetic nerves include a substantial portion of the proprioceptive reflex system. There is no reason to assume that the quality of reflexes elicited by stimulation of proprioceptive receptors in the muscle is determined by the path of the peripheral components of the reflex arc involved. At the same time the experimental data which indicate that the sympathetic system is a factor in the production of muscle tonus, also indicate that the component of tonus mediated through the sympathetic system is essentially plastic in quality. This is probably not the only factor in plastic tonus. Dr. Kuntz and Dr. Kerper again refer to their finding that after the elimination of the sympathetic nerve supply of a muscle it is possible that no postural defect may be detected by direct observation or palpation in the waking animal, although a reduction in tonus in the absence of voluntary contraction may be found by the method of measurement described by them. Moreover, the tonus curves of the quadriceps muscle deprived of its sympathetic nerve supply do not indicate complete absence of tonus in the resting muscle. The tonus which persists in this muscle in the absence of contractile effort, is essentially postural and probably includes a component which is plastic in quality.

Abstracts from Current Medical Literature.

MORPHOLOGY.

The Superficial Venous System of the Lower Extremity.

CHARLES KOSINSKI (*Journal of Anatomy*, January, 1926) discusses varicosis of the superficial venous system of the lower extremity with special reference to the part played in its causation by the small saphenous vein and also the hereditary tendency which several writers state accounts for a larger percentage of varices. He holds that this hereditary factor may be expressed in the modes of arrangement of the superficial veins.

The Innervation of the Heart.

H. H. WOOLLARD (*Journal of Anatomy*, July, 1926) reviews the literature on the subject of the innervation of the heart and gives a detailed account with figures of his findings. The object of his investigation was to determine if possible the distribution of the vagus and sympathetic fibres of the heart and an attempt was made to see if a combined morphological and experimental investigation of a doubly innervated organ such as the heart would add to the knowledge of the manner in which such a type of innervation is arranged. By combining the methylene blue *intra vitam* staining method with the process of clearing used in the Spalteholz method the writer was able to make transparent preparations of the whole surface of the heart and thus investigate the distribution of the cardiac ganglia and nerve trunks. When the ultimate distribution of the nerve fibres of the vagus and the sympathetic nerves was followed it was found that they became indistinguishable from one another and this made the interpretation of degeneration experiments difficult. Some evidence was brought forward to suggest that the auricles and atro-ventricular bundles are supplied by both parasympathetic and sympathetic fibres, while ventricular muscle is supplied by sympathetic fibres only. The fibres spin a plexus about the muscle cell and ultimately enter the protoplasm of the muscle and run in the protoplasmic connexions of the muscle. Small endings, often perinuclear in position, are given off inside the muscle cell. In the auricles fibres from both systems appear to end intramuscularly. Nerve endings on the coronary vessels are also described.

The Frenula Valvulæ Coli.

A. H. RUTHERFORD (*Journal of Anatomy*, July, 1926) records observations made on several *post mortem* specimens of the bowel in the region of the ileo-caecal junction. The specimens examined were from the monkey immediately after death and from man within twenty-four hours of death. From an examination of the specimens both macroscopically and microscopically the writer concludes that the human frenula are in reality the caeco-colic sphincter in the human subject and that the human caeco-colic sphincter is functionally active.

The Projection of the Retina in the Primary Optic Neurone in Monkeys.

B. BROUER AND W. P. C. ZEEMAN (*Brain*, Volume XLIX., Part I., 1926) take up the problem of cerebral organization of vision and attempt an analysis of the conditions in the external geniculate body, in the occipital lobe and in the tracts between them. The article contains, in addition to a short introduction and a reference to earlier publications, a description of secondary degeneration after sixteen experimental lesions of the retina in monkeys. The authors studied by Marchi's method sections of the brain after extirpation of one eye, after partial lesions in the periphery of the retina and after operations on the upper and lower halves of the macula. This study gives an indication of the localization of the different quadrants of the retina in the primary optic neurones. The authors especially draw attention to the work done on the projection of the retina in the external geniculate body. In the latter part of the paper they discuss their results in connexion with the literature on the subject. Finally they indicate the significance of their results and their effect on the analysis of the relations in the second optic neurone, that is in the central optic radiations and in the calcarine cortex.

The Retina and Lateral Geniculate Body in the Primates.

H. H. WOOLLARD (*Brain*, Volume XLIX., Part I., 1926) gives a preliminary report on work being done in the Department of Anatomy at University College, London, with the object of investigating the anatomical details of the organs of discriminative sensibility in the primate series. The significance and importance of this aspect of man's evolution has been pointed out by Elliott Smith in his many contributions to the subject. This communication concerns the differentiation of the retina and the concurrent changes that are to be found in the lateral geniculate body in a series of closely related animals forming an ascending series.

The Function of the Right Hemisphere of the Brain in Relation to the Left in Speech, Music and in Calculation.

S. E. HENSCHEN (*Brain*, Volume XLIX., Part I., 1926) draws attention to the predominant and exclusive rôle played by the left hemisphere of the brain in speech, music and calculation and comments on the relation of these functions to right-handedness. He also discusses the question of asymmetry and states that no constant predominance of one-half of the body over the other has been proved by exact observations in quadrupeds. Interesting observations are recorded with regard to the relation of left-handedness and inferior intelligence. The writer comes to the conclusion that all the facts seem to be in favour of the theory of a primary organization of the brain as the original cause of the predominance of the left side of the brain and states that in Nature there seems to be a law that in order to attain full development of higher faculties originally distributed over two organs a concentration of the capacity in only one of them is necessary and that development in Nature always depends on the differentiation of a preexisting faculty.

Asymmetry in Hermaphrodite Pigs.

JOHN R. BAKER (*Journal of Anatomy*, July, 1926) describes two new true hermaphrodite pigs and brings together all he has been able to discover about asymmetry in the literature of such monstrosities. He summarizes his results as follows: (i) The left gonad of hermaphrodite pigs is usually an ovary or ovotestis, while the right gonad is usually a testis or ovotestis. (ii.) The ovarian part of ovotestes is always in the same place in relation to the testis and adjacent parts. It is situated ventro-anteromedially to the testis, near the right angle bend in the epididymis. (iii.) The epididymis, *vas deferens*, *vesiculæ seminales*, pampiniform plexus and Fallopian tubes tend to show an asymmetry corresponding with that of the gonads. (iv.) Some influence other than the gonad-hormones affects the development of the accessory reproductive organs. This influence is swamped by either gonad-hormone acting alone, but shows itself when both testicular and ovarian hormones are present to counterbalance one another.

MORBID ANATOMY.

Cervicitis.

G. L. MOENCH (*The American Journal of Obstetrics and Gynecology*, April, 1926) discusses cervicitis, erosion, laceration *et cetera* from the point of view of their pathological ætiology. The epithelial lining of the genital tract in the female is described both in the developmental and adult stages. Pseudoerosion is then dealt with and this is described as the normal columnar epithelium of the cervical canal growing out beyond the level of the internal *os*. Sometimes this is spoken of as congenital erosion, for later this epithelium becomes replaced by squamous epithelium and in this connexion it would appear to be borne out by the fact that the injection of ovarian extract into the newborn causes an epithelial hyperplasia in the vagina. Syphilis and tuberculosis also affect the cervix, but their nature is similar to that occurring in lesions elsewhere. There is a specific gonococcal cervicitis and this is characterized by the formation of pointed condylomata similar to those occurring on the skin in this disease. This form of lesion is rare and the author definitely states that all mucopurulent discharges from the cervix

cannot be called gonococcal. The histological changes in infections of the cervix occur mainly in the stroma with polymorphonuclear invasion in the more acute lesions and lymphocytic infiltration in the chronic type. The author states that plasma cells can no longer be thought characteristic of gonococcal infections. Ectropion usually follows acute infections and is comparable to ectropion of the urethra. This is to be distinguished from cervical erosion which is the direct result of an inflammatory lesion, and in it the mucosa is denuded to be later covered by columnar epithelium and later still this columnar epithelium is converted into squamous epithelium. As the erosion heals, it rapidly becomes replaced by squamous epithelium and as it advances, it undermines the columnar epithelium and may extend into the glands and histologically may simulate malignant disease. In laceration the exposed epithelium is columnar and gives the appearance of an erosion, but later the squamous epithelium may grow further into the cervical canal than is usual.

Fat Necrosis in the Breast.

GEOFFREY HADFIELD (*The British Journal of Surgery*, April, 1926) describes fat necrosis in the breast and gives an account of a case. Inflammatory processes of the breast are frequently encountered in the routine examination of excised breast tissue. Generally the gross and microscopical characters are similar to those found in inflamed fat in other situations. When a pendulous breast suffers trauma, a lump may appear and may resemble carcinoma clinically in regard to its hardness and adherence to the skin. Histological examination of tissue showed that the nodule was composed of large swollen cells with foamy cytoplasm separating and distorting large fat globules. There is a slight fibroblastic reaction, but the lesion is almost bloodless. A few of the fat globules are filled by faintly staining material and in some areas clefts and spaces are seen. Macroscopically the specimen consisted of fat with small nodules which were roughly circular in outline and measured about twelve and a half millimetres (half an inch) in diameter; chalky-white fasciculi were found radiating from the nodules.

Oxycephaly.

DAVID M. GREIG (*Edinburgh Medical Journal*, April, May and June, 1926), in a communication running through three numbers, discusses oxycephaly. He says that its occurrence is rare and only a few instances are seen by any one individual. True oxycephaly is recognizable at birth and is due to general cranio-facial synostosis and is associated sometimes with other congenital anomalies. Three cases are reported and the condition in each instance is illustrated with photographs and radiograms and skull measurements are appended. In this condition the skull rises to the bregma to a blunted apex, the orbital cavities are seen to be very shallow, while on either side the temporal regions bulge beyond the zygomata. There is no trace of coronal suture. The body of the mandible is strong and heavily formed and the whole of the facial bones are small and atrophic. Full description of the *norma basalis, verticalis et cetera* with the finer details of the abnormalities are discussed. Both internally and externally the bony surfaces are smooth and do not present the irregularities associated with inflammatory processes. The skull is thin everywhere and there is a striking absence of diploe and the capacity of the cranium in the case under discussion is 1,620 cubic centimetres. True oxycephaly is compared with delayed oxycephaly and contrasted with false oxycephaly. In the latter the height of the skull is a misleading feature and its occurrence is neither congenital nor hereditary and some sutures only are affected. The facial characteristics of true oxycephaly together with the small pharynx lead to a condition of snuffles.

Chorionic Carcinoma.

R. M. HANDFIELD-JONES (*The British Journal of Surgery*, April, 1926) describes chorionic carcinoma of the testis and reports a new case. The writer quotes Sanger as being the first to recognize the uterine tumour in 1889. Malassez and Monod in 1878 reported a condition which they originally called *sarcome angioplastique*, but which later was recognized as being a replica of these tumours of the uterus. The naked eye appearance of chorionic carcinoma produces a typical picture according to the elements present. The microscopical picture consists of a syncytium with Langhans's layer, also of chorionic wandering cells and masses of fibrin and red cells. The syncytium is in intimate relation with the blood capillaries and is seen eroding them and the chorionic wandering cells appear to be the forerunners of the eroding process. As regards the blood and fibrin they are highly suggestive of the diagnosis. Metastases are widespread and it is usual for the secondary deposits to manifest the features of the primary growth and often examination of the former puts the origin of the tumour beyond doubt. Generally it is accepted that the tumour is derived from some elements in a teratoma. Activity of a male breast may be occasioned by the presence of one of these tumours and the presence of thyreoid tissue in a teratoma may produce a state of hyperthyreoidism.

Inflammation of the Thyreoid Gland.

JOHN TAYLOR WATKINS (*Annals of Clinical Medicine*, February, 1926) discusses inflammation occurring in the thyreoid gland. Inflammation is infrequent, though there are many references in older literature to thyreoiditis. The existence of this condition is based on findings of localized collection of lymphocytes either with or without germ centres, but at present it is regarded as belonging to a thymico-lymphatic constitution. The thyreoid gland appears to be relatively immune to bacterial invasion. Non-suppurative lesions are thought to occur in certain conditions, while suppurative thyreoiditis has been seen by the author during the course of or during convalescence from Vincent's or Ludwig's angina, typhoid fever *et cetera*. Interstitial thyreoiditis of the non-suppurative variety occurs in those glands manifesting simple hypertrophy and parenchymatous hypertrophy. In the "woody" thyreoid the fibrosis is very prominent and infiltrates the surrounding structures. Tuberculosis in some instances is thought to be a primary condition, while the occurrence of syphilis is very rare. Varying grades of hypofunction and even myxœdema may follow any of the forms of inflammation.

Malignant Neoplasm of the Thyreoid Gland.

WALTER M. SIMPSON (*Annals of Clinical Medicine*, February, 1926) discusses malignant neoplasms of the thyreoid gland. With regard to the carcinomata he thinks that these epithelial tumours fall into groups in a similar fashion as do carcinoma of other organs. In all 1,449 examinations of thyreoid tissue were made and the changes in 159 of these were diagnosed as being due to exophthalmic goitre. In none of them was there any evidence of neoplasm. From comparative figures of its occurrence it appears that the condition is more frequent than is believed. Of the malignant tumours 72% occurred in females and 28% in males and three-fourths of the patients were over thirty-five years of age. Analysis of the case records reveals that in a large number the diagnosis was not made clinically and that many tumours were associated with adenomata; a number of these were "toxic adenomata." In every case there was a history of a previous goitre which was usually first observed at puberty. Rapid growth, pressure effects, pain and other nervous symptoms characterized the tumour growth which may also originate in aberrant thyreoid tissue. Metastases are widespread. Medullary carcinoma occurred in 36% of cases and in these the clinical diagnosis was correct. Of the tumours 60% were adeno-carcinomata with its predominant acinar arrangement. Pale staining colloid or more often no colloid was present and the tumour was thought to have originated in an adenoma and not in the author's experience in any fœtal adenomata. None of the diagnoses were made before operation. Scirrhous carcinoma was rare and comprised but 2% of the total. A sarcoma occurred once to every carcinoma and in this series all occurred in adults past middle life. This was a very noticeable feature. In the sarcomata all varieties of cell were encountered.

British Medical Association News.

MEDICO-POLITICAL.

MEETING OF THE FEDERAL COMMITTEE.

A MEETING OF THE FEDERAL COMMITTEE OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA was held on August 25 and 26, 1926, at the B.M.A. Building, 30-34, Elizabeth Street, Sydney, SIR GEORGE A. SYME, K.B.E., Chairman, in the chair.

Representatives.

The following representatives of the Branches were present:

New South Wales Branch: Dr. R. H. Todd, Dr. J. Adam Dick, C.M.G.
Victorian Branch: Sir George A. Syme, K.B.E., Dr. R. H. Fetherston.
Queensland Branch: Dr. W. N. Robertson, C.B.E., Dr. J. Lockhart Gibson.
South Australian Branch: Dr. H. S. Newland, C.B.E., D.S.O., Dr. Bronte Smeaton.
Tasmanian Branch: Dr. D. H. E. Lines.

Apologies for non-attendance were received from Dr. Brettingham Moore who had been appointed representative of the Tasmanian Branch in the place of Dr. Gregory Sprott and from Dr. D. D. Paton, the representative of the Western Australian Branch. Dr. Paton appointed Dr. R. H. Todd to act as proxy.

Financial Statement.

The financial statement of the Federal Committee duly audited by the Honorary Auditor was received and adopted.

Medical Officers' Relief Fund (Federal).

The Trustees of the Medical Officers' Relief Fund forwarded a letter explaining the position of the fund, together with a balance sheet and revenue account for the twelve months ended June 30, 1926. It was resolved that the thanks of the Committee be communicated to the Trustees, that the financial statement be received and that it be published in THE MEDICAL JOURNAL OF AUSTRALIA. A discussion took place on the ultimate use of the moneys of the fund. It was noted that the terms of the trust provided that "the fund shall be used in assisting medical officers who have been disabled and the dependants of those who have died." It was anticipated that the assistance of the dependants of the medical practitioners who had lost their lives in the war, would possibly exhaust the whole fund in the course of time.

Business of the Committee.

A communication from the Western Australian Branch was read dealing with the difficulties experienced by the Branch in considering the matters referred to it by the Federal Committee shortly before the holding of meetings. It was explained that according to the constitution of the Committee fifty-six days' notice of meeting had to be given. As a rule a preliminary business paper was issued at the time when the notice convening the meeting was circularized to the members. At times, however, further motions were submitted after the notice papers had been issued. The suggestion that a special expedient be adopted in order to expedite the transmission of the later notices of motion to the Western Australian Branch was considered and accepted.

Post-Graduate Work in London.

A letter from the Western Australian Branch was read accompanied by letters from the authorities in Australia House in regard to the Fellowship of Medicine and the Post-Graduate Medical Association. It had been stated that full use of the facilities provided by these two bodies was not being made by Australian practitioners visiting London. Expressions of opinion concerning the value of the post-graduate arrangements in London from various prominent practitioners were submitted. Some of the practitioners extolled the value of the post-graduate training in terms of high praise, while others expressed the opinion that the training was of relatively small value in regard to the specialties, although it was satisfactory in regard to general work. It was resolved that the attention of the Western Australian Branch should be directed to the action taken by the Federal Committee at its last meeting in February, 1926 (see THE MEDICAL JOURNAL OF AUSTRALIA, February 20, 1926, page 220).

British Medical Association Scholarships and Grants.

It was reported that an application by an Australian practitioner for a British Medical Association scholarship had been received and that the application, together with recommendations, had been forwarded to London. Unfortunately the number of applications received by the Council in London had been large and the Australian graduate had been unsuccessful in obtaining the scholarship desired.

Professional Secrecy.

A letter from the Western Australian Branch was read drawing attention to the fact that the Federal Taxation Department had demanded that the nature of the particular illness or operation should be stated by the taxpayer when claiming deduction of the amount paid by him to medical practitioner, public or private hospital, nurse or chemist, as provided by the Act. It was resolved that a communication should be addressed to the Federal Treasurer containing an expression of opinion of the Federal Committee that the nature of an illness should not be required to be disclosed for the purpose of the substantiation of claims for exemption from taxation.

Medical Ethics.

A letter was read from the Queensland Branch together with correspondence dealing with a charge of unethical conduct on the part of a member of the Branch. The material submitted comprised an *ex parte* statement in the course of which the complainant stated that one medical practitioner had advised his patient to enter a public hospital and the patient had instead entered a private hospital under another practitioner's care. The question submitted by the Queensland Branch to the Federal Committee was whether advising a patient to enter a public hospital was tantamount to relinquishing the case.

Objection was taken to the proposal that the Federal Committee should endeavour to adjudicate on an ethical charge. It was pointed out that the question submitted by the Queensland Branch was not one on a hypothetical case, but had direct reference to the specific complaint which had been investigated by the Branch, and could be regarded as an interpretation of the Principles of Medical Ethics adopted by the Federal Committee.

It was resolved that the question whether advising a patient to enter a public hospital was tantamount to relinquishing the case did not admit of an answer yes or no and that each case should be determined on its merits.

Remuneration of Ships' Surgeons.

A communication was read from the Victorian Branch enclosing a letter and a cutting from a newspaper in connexion with the remuneration and conditions of service of ships' surgeons. This matter had been discussed by the Federal Committee in July, 1922 (see THE MEDICAL JOURNAL OF AUSTRALIA, July 29, 1922, page 139, and February 24, 1923, page 218). The arrangements made at that time with the Australasian Steamship Owners' Federation had proved satisfactory as far as ships registered in Australia were concerned. Ships' surgeons carried on vessels in accordance with the provisions of the *Navigation Act* received not less than £25 per month and were entitled to charge a fee of five shillings for attendance on first class or second class passengers. It was stated that ships registered elsewhere and trading in Australian waters at times did not carry a ship's surgeon when the distance between the ports of call was over 600 miles and that other ships were paying their surgeons inadequate sums. The arguments in favour of surgeons and nurses being carried on board all passenger vessels was set out in the article referred to above. It was resolved to bring the

FEDERAL COMMITTEE OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.

STATEMENT OF RECEIPTS AND PAYMENTS FOR SIX MONTHS ENDED JUNE 30, 1926.

RECEIPTS.	£ s. d.	£ s. d.
To Balance at 31st December, 1925—		
Union Bank of Australia, Ltd.	115 1 4	
Government Savings Bank of New South Wales	534 16 4	
Cash on hand	2 2 6	
		652 0 2
„ Interest—		
Government Savings Bank of New South Wales		18 3 7
		£670 3 9

PAYMENTS.	£ s. d.	£ s. d.
By Travelling Expenses		38 13 4
„ Sundry Expenses		139 2 4
„ Balance at June 30, 1926—		
Government Savings Bank of New South Wales	£502 19 11	
Cash on hand	1 6 2	
	504 6 1	
Less Union Bank of Australia, Ltd., Overdraft	11 18 0	
		492 8 1
		£670 3 9

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION) ACCUMULATED FUNDS ACCOUNT.

RECEIPTS.	£ s. d.	£ s. d.
To Balance at December 31, 1925—		
Government Savings Bank of New South Wales	278 8 5	
„ Interest—		
Government Savings Bank of New South Wales	11 8 9	
	£289 17 2	

PAYMENTS.	£ s. d.	£ s. d.
By Clerical Assistance		7 10 0
„ Balance at June 30, 1926—		
Government Savings Bank of New South Wales		282 7 2
		£289 17 2

(Sgd.) ROBERT H. TODD,
Honorary Secretary.

Audited and found correct—
(Sgd.) W. H. CRAGO,
Honorary Auditor.

Sydney, August 24, 1926.

MEDICAL OFFICERS' RELIEF FUND (FEDERAL).

Balance Sheet as at June 30, 1926.

LIABILITIES.	£ s. d.	£ s. d.
Accumulated Revenue Account—		
Balance at June 30, 1926 ..	11,477 16 11	
Add Balance from Revenue Account for year ended June 30, 1926	81 5 8	
		11,559 2 7
		£11,559 2 7

ASSETS.	£ s. d.	£ s. d.
Investments—		
6% 1930 Commonwealth Treasury Bonds	1,000 0 0	
5% 1948 Commonwealth Treasury Bonds	250 0 0	
British Medical Association, New South Wales Branch Building Account Debentures ..	300 0 0	
		1,550 0 0
Loans Bearing Interest		8,618 10 0
Fixed Deposit		1,000 0 0
Amount in Hands of Local Committee, Queensland		5 0 0
Bank of New South Wales—		
Current Account		385 12 7
		£11,559 2 7

Revenue Account for the Twelve Months Ended June 30, 1926.

	£ s. d.	£ s. d.
To Benefactions	424 10 0	
„ Expenses	22 10 10	
„ Balance to Accumulated Revenue Account ..	81 5 8	
	£528 6 6	

	£ s. d.	£ s. d.
By Interest on Investments and Loans	545 14 9	
Less Interest on Bank Overdraft ..	17 8 3	
		528 6 6
		£528 6 6

We have examined the books, vouchers and securities of the Medical Officers' Relief Fund (Federal) and we certify that in our opinion the foregoing Balance Sheet is properly drawn up so as to exhibit a true and correct view of the financial position of the fund as at June 30, 1926.

COATES, CUNNINGHAM & COMPANY,
Public Accountants.

Sydney, August 9, 1926.

matter to the attention of the Minister for Health. In view of the fact that the conditions of employment of surgeons on board New Zealand ships was stated to be unsatisfactory, the Committee determined to communicate with the New Zealand Branch and to institute further inquiries.

Australasian Pharmaceutical Conference.

A communication was read from the Honorary General Secretary of the Australasian Pharmaceutical Conference inviting the Federal Committee to meet the members of the Federal Pharmaceutical Council for the purpose of discussing the problems in connexion with national health insurance. In view of the fact that very little information was available the Committee resolved to leave the matter in the hands of the Chairman and Honorary Secretary.

Australasian Medical Congress (British Medical Association).

Suggested Section of Anæsthesia.

A letter from the Honorary General Secretary of the Second Session of Australasian Medical Congress (British Medical Association), Dunedin, 1927, enclosing a letter from the Anesthesia Research Society of Ohio, United States of America, in which the writer suggested that a section of anæsthesia should be established at the Congress. The procedure in regard to the formation of sections is governed by Regulation 3 (*b*) (5). It is recognized that it was too late for the Executive Committee to form a further section and that the Federal Committee had no discretion in the matter.

Honorary Members.

In accordance with the regulations the Executive Committee had forwarded the names of persons nominated for election as honorary members. It was resolved that the following be appointed honorary members of the Second Session of Congress: Baron Kitasato (Tokyo); Professor K. Shiga (Tokyo); L. B. Bull, D.V.Sc. (Adelaide); Professor T. Brailsford Robertson, D.Sc. (Adelaide); Lieutenant-Colonel H. M. Hays, M.D. (New York); Surgeon Commander B. J. Dudley (H.M.A.S. *Dunedin*).

Vice-Presidents and Patrons.

It was announced that Dr. A. C. Purchas had intimated since the last meeting of the Federal Committee that he was pleased to accept the office of Vice-President. The Honorary Secretary informed the Committee that he had forwarded the names of the patrons and vice-presidents to the Honorary General Secretary in Dunedin. It was announced that His Excellency Baron Somers, K.C.M.G., D.S.O., M.C., the Governor of Victoria, would become patron in the place of the Earl of Stradbroke. It was further determined that the Acting Governor of Queensland be invited to accept the position of patron.

Date of Congress.

A telegram was read from the Honorary General Secretary of the Second Session of the Australasian Medical Congress intimating to the Federal Committee that the date of the commencement of the Session had been postponed from February 2 to February 3, 1927.

Incorporation of Overseas Branches.

Correspondence with the Queensland Branch was read and it was noted that the Branch had become incorporated on May 3, 1926, under the Memorandum and Articles of Association approved by the Council of the British Medical Association.

In regard to a letter that had been received from the Western Australian Branch, the Honorary Secretary announced that the by-laws which were being drafted for the New South Wales Branch were not quite complete. These by-laws were still in the hands of the solicitors of the Branch. It was hoped that these by-laws might prove useful to other Branches seeking incorporation as companies not for profit.

Industrial Hygiene Contracts.

The Honorary Secretary announced that the draft Memorandum of Agreement between the industrial medical officer and company or firm which had been adopted by the Federal Committee at its last meeting (see THE MEDICAL JOURNAL OF AUSTRALIA, February 20, 1926, page 222) had been circularized to the Branches with a request that suggestions be made concerning the remuneration of industrial medical officers. Replies had been received from the Western Australian Branch, the Tasmanian Branch, the Queensland Branch, the New South Wales Branch and the Victorian Branch. The Western Australian Branch had found that the work was being conducted in that State by the Government Medical Department under the *Factories Act*. No private medical practitioners were undertaking industrial hygiene work. Neither the Tasmanian Branch nor the Queensland Branch had sufficient experience to justify them in making any suggestion concerning remuneration. The New South Wales Branch suggested the following rates of remuneration: £1 11s. 6d. per hour or £3 3s. for a half-day of three hours, £5 5s. for a whole day of six hours. The Victorian Branch notified that the following rates had been adopted: £1 1s. per attendance of one hour or part of an hour per day; ordinary fees for visits to the factory outside specified hours; if frequent attendances were required a minimum salary to be paid should be subject to the approval of the Council. If attendance each day were required a minimum salary of £250 a year should be paid.

It was thought that few industrial firms would be prepared to engage whole-time or half-time medical officers and that it might be difficult to persuade firms to carry out the principles of industrial hygiene in a thorough manner. For this reason it might be inadvisable to prescribe inelastic terms. In Victoria even large industrial undertakings were satisfied with a visit of a half an hour to one hour a day and the majority did not pay more than one guinea per visit. The work was being done by general practitioners. As this department of medical work was developed, it was anticipated that practitioners would make a special study of it and these men would command higher fees. Under certain circumstances higher rates should be paid. For example, in some trades the medical officer was required to watch the workers for signs of industrial poisoning. It would be necessary to pay a higher rate when this was required. It was doubtful whether at this stage uniformity of conditions in the several States of Australia could be secured. After a full discussion it was resolved:

> That the rate of remuneration for industrial medical officers be not less than one guinea per hour or part of an hour.

Dichotomy.

The Honorary Secretary reported that in accordance with the instructions given at the last meeting he had forwarded to the several Branches a copy of the Memorandum addressed by the Council of the New South Wales Branch to its members on the relative position of the medical attendant and the consultant, operating surgeon, pathologist, bacteriologist, radiologist or other specialist in respect to the patient or the patient's representative in the matter of fees (see THE MEDICAL JOURNAL OF AUSTRALIA, February 20, 1926, page 223). A letter had been received from the Queensland Branch requesting the Federal Committee to define the term dichotomy. Several examples of actions that might be regarded as dichotomy were quoted and the letter concluded with the following paragraph:

> It is our opinion that an account should state clearly the amount paid to each medical practitioner concerned and that any money paid by one practitioner to another, the exact amount of which is not stated on an account to the patient, is dichotomy.

While the majority of the members approved of the suggestion that an account rendered to a patient by a practitioner for services carried out by himself and one or more of his colleagues should contain details of these services, it is recognized that under certain circumstances no objection could be taken to an account not detailed in this manner. Fee splitting or dichotomy was obviously a wrongful deed, whereas the neglect to intimate to the

patient the proportions of the fees paid that would be handed over to those assisting or performing an operation was not necessarily an indication of dishonesty. The offence had been defined in paragraph 10 of the Principles of Medical Ethics adopted by the Federal Committee as follows: "That it is unethical for a member . . . . to divide fees with or give commissions to medical practitioners or laymen." The opinion was expressed that it was improper for a surgeon to pay to a general practitioner fees in excess of the usual fees paid under the obtaining circumstances. The Committee decided to reply to the Queensland Branch that it did not necessarily follow that an account set out in the way indicated in the letter constituted dichotomy. The Committee was disinclined to adopt any further definition than was included in the Principles of Medical Ethics.

Royal Commission on Health.

A letter was read from the Prime Minister to the effect that the request of the Federal Committee that an opportunity should be given it of considering any proposals for legislation arising from the report of the Royal Commission on Health before they were submitted to Parliament, would receive consideration. It was noted that a conference had been held between representatives of the Federal Government and the State Governments concerning the several recommendations included in the report. As no further communication had been received from the Prime Minister it was resolved to approach both him and the Minister for Health on the matter.

In response to a common letter addressed to the Branches, the Victorian Branch had replied "approving generally of the report and suggesting certain addenda." In Division I.—Ill Health in the Commonwealth, recommendation (3) (see THE MEDICAL JOURNAL OF AUSTRALIA, January 16, 1926, page 57) should be amplified by the addition of the words: "For the purposes required by the Commonwealth Statistician uniform certificate forms should be furnished to the State Health Department." It was pointed out that it was held to be desirable that medical practitioners should be required to forward such statistical information as might be required to the Health Departments of their respective States whose duty it would be to forward this information to the Commonwealth authority. The use of uniform certificates would not only render the information of greater value for statistical purposes, but it would also lessen the burden of the private practitioner. The Committee resolved to add the words to the recommendation.

The Victorian Branch proposed in Division III.—Co-operation of Commonwealth and State Health authorities, to add after recommendation (6) (see page 62):

> (7) That the State Governments be asked to request the Commonwealth Government to take over general powers to legislate on health.

It was pointed out that the Commissioners had dealt with the question of the advisability of asking the Commonwealth Government to secure powers to legislate on health. The Commissioners had not recommended the Commonwealth Government to obtain general powers through a referendum. On the other hand it was recognized that if the States voluntarily surrendered their powers, the Commonwealth Government would be in a position to undertake the duties. The opinion was expressed that it was extremely doubtful whether the States would be prepared to relinquish these rights voluntarily. The motion for the inclusion of the new recommendation was lost.

The third proposal of the Victorian Branch was an addition to recommendation (4) in Division V.—Venereal Disease. Recommendation (4) reads as follows: "More clinics should be established in the larger centres and more widely distributed in each centre" (see page 67). The proposal was to add the words: "The medical staffs should receive adequate remuneration." The motion was carried. The last proposal of the Victorian Branch was under Division XI.—The Relationship which should Exist Between Public Health Authorities and Medical Practitioners (see page 78). It was proposed to add to recommendation (3) the words: "And strongly approves that disciplinary powers should be taken under principal act of registration." It was pointed out that the words "and strongly approves" were not needed. The addition as amended was approved.

The reply from the New South Wales Branch was to the effect that the Council had considered in detail those portions of the report which it regarded as of immediate interest to medical practitioners. Approval was given to the model scheme in Division IV., to Division XI. and to Division XIII. The Council had hesitation in approving of recommendation (13) (*b*) in Division V.—Venereal Disease (see page 67). This recommendation reads as follows:

> When the Commonwealth passes legislation in the exercise of its constitutional powers regarding marriage and divorce, it should include clauses dealing with venereal disease, especially that
>
> (*a*) . . . . .
>
> (*b*) A clause providing that no marriage should be allowed unless the parties make declaration that they are not suffering from any contagious or infectious disease.

The reply from the Western Australian Branch contained a general approval of the report with comments and suggested alteration. Under Division II.—Medical Services in the Commonwealth—(*a*) Medical Services of Civil Department (see page 58), the Western Australian Branch wished to emphasize the need of a much stricter control of migrants for Australia and their families. It was pointed out that the appointment of Dr. C. L. Park as Commonwealth Medical Officer attached to the High Commissioner's Office in London was an important step toward the achievement of this object. If Sir Neville Howse were sent to London, he would do his utmost to support the endeavour to prevent unhealthy persons from migrating to Australia. In these circumstances it was thought that it would be sufficient if the views of the Federal Committee were explained to the responsible authority.

The second matter with which the Western Australian Branch dealt, was in connexion with the establishment of laboratories for diagnostic purposes. The Branch suggested that these laboratories should be under the control of the States, save those established by the Federal Department of Health for the purpose of conducting special investigations of problems affecting the Commonwealth as a whole. In the course of a discussion it was pointed out that the Royal Commissioners had given this matter serious consideration and had recommended that legislation should be passed by the Federal Parliament. The reference of the Royal Commission had been to inquire into and report upon public health as a matter for legislation and administration by the Commonwealth, in conjunction with the States where necessary. The Commissioners had not been asked to examine the question of State control, save in relation to any action undertaken by the Federal authority. The Federal Committee was not in accord with the opinion of the Western Australian Branch, in view of the fact that the work conducted in the Commonwealth laboratories in the several States had proved to be excellent.

The opinion was expressed in the letter from the Western Australian Branch that the recommendation (3) (*a*) under Division VII.—Maternity, was impracticable in certain outlying districts where there was no doctor. The wording of the recommendation was as follows:

> The *Maternity Allowance Act* should be amended to provide that the application for the allowance should be made at least five months before the date of the expected childbirth and that the allowance be not paid unless a medical certificate be produced to the effect that the mother has had prenatal supervision.

Dr. R. H. Fetherston stated that this matter had received attention by Dr. Marshall Allen, the Director of Obstetrical Research in Victoria and that means could be devised to meet the exigencies of outlying centres. It was usually possible to secure some supervision by a nurse if no doctor were available. Other speakers arrived at the con-

clusion that if the recommendation could not be applied in exceptional circumstances, provision could be made in an amending bill. It was therefore resolved that the comment of the Western Australian Branch be noted and that if it were found that the proposed condition could not be applied in certain districts, a clause could be inserted to exclude women in those districts from its operation.

The last comment of the Western Australian Branch was a disapproval of the recommendation (1) under Division XI.—The Relationship which should Exist Between Public Health Authorities and Medical Practitioners. This recommendation dealt with the provision of statistics by medical practitioners to the Commonwealth Statistician. The Western Australian Branch considered that the Commonwealth Statistician should obtain all the statistics he required through the State Department of Health and should not have direct dealings with medical practitioners. It was explained that the Royal Commissioners had chosen the other method in order to obviate certain difficulties that might arise. It was desirable to insure that the statistical information sought should become available and for this purpose legislation might be needed. The Federal Government had no power to impose duties on the State Departments. If a State Department was not interested in the matter, the Commonwealth Statistician might not obtain the information he required. It was resolved to explain the difficulties to the Western Australian Branch.

Uniform Conditions of Medical Services in Public Hospitals.

At the last meeting of the Federal Committee it was resolved to ask the Branches to supply full statements concerning the systems existing in the various hospitals in their respective States. A considerable amount of information was presented to the Committee.

It was resolved that a subcommittee consisting of the Chairman, Sir George Syme, and Dr. Fetherston, with power to coopt other members should collate the material and present a statement to the next meeting of the Federal Committee.

Medical Registration.

Correspondence was read from the Queensland Branch in regard to the inadvisability of the transfer of the powers to legislate on matters relating to the registration of medical practitioners to the Commonwealth Parliament. A short discussion ensued. In view of the recommendation of the Royal Commission on the subject of uniformity of these laws, it was decided to take no further action.

Salaries of Commonwealth Medical Officers.

Correspondence was read from which it transpired that the Chairman had been asked to take action in connexion with an increase of salaries of the Commonwealth medical officers. In view of the policy laid down in the Hone-Newland report in connexion with the salaries of medical officers in the Department of Health of the Commonwealth (see THE MEDICAL JOURNAL OF AUSTRALIA, February 28, 1925, page 223), the Chairman, Sir George Syme, had given favourable consideration to the request of the medical officers for support of their claims for increased rates of remuneration. The matter of the salaries of all the medical officers with the exception of the Director-General was governed by an award. Permission had been sought and obtained from the several members of the Federal Committee for Sir George Syme and Dr. R. H. Fetherston to interview the Minister for Health on behalf of the medical officers. This had been done and Sir George Syme had also given evidence before the arbitrator. He had pointed out that the Federal Committee had recommended that the Director-General of Health should be paid a salary of at least £2,000 to £2,500 a year and that the salaries of the medical members of his staff should be in proportion. Chief Commonwealth Medical Officers in the several States should be paid at least £1,500 a year. It had further been pointed out that the salaries of the medical officers holding positions at the present time should be increased and that the higher rate should be paid to all newly appointed medical officers. The arbitrator had been favourably impressed and a general increase of approximately 16% had been granted. The Committee thanked Sir George Syme for his timely action.

Confidential Information to Insurance Companies.

A letter was read from the Victorian Branch requesting the Federal Committee to consider a series of resolutions that had been adopted by the Council of the Branch on June 14, 1926. According to these resolutions it would not be unethical for a practitioner to give relevant information to a life insurance company, provided that the proponent had signed a statement consenting to the giving of this information. The wording of the statement approved by the Council of the Victorian Branch was appended.

The letter had been sent to the several Branches and replies had been received from the Western Australian Branch and the Tasmanian Branch approving of the resolution. The New South Wales Branch had disapproved. Dr. R. H. Todd in explanation of the disapproval of his Branch, pointed out that the procedure followed by life insurance companies was usually to insert a clause of this nature in the form which proponents were required to sign. It was doubtful whether the clause was read. The real significance was certainly not understood by the majority of proponents. A little consideration of the matter revealed the fact that this clause could be used by insurance companies for the purpose of repudiating claims. It was becoming more frequent for companies to issue policies without a previous medical examination. Claims made under these policies were not paid if evidence were forthcoming that the proponents had not been in good health at the time of application. The object of this clause was to enable the companies to obtain this evidence. Dr. Todd therefore moved that the Federal Committee disapproved of the resolutions of the Council of the Victorian Branch. The motion was seconded by Dr. J. Adam Dick and was carried.

Friendly Society Lodge Practice.

A letter from the Queensland Branch was read, inviting the Federal Committee to consider the proposal to devise and adopt uniform conditions of friendly society lodge practice throughout Australia. The Queensland Branch suggested that the Common Form of Agreement of the New South Wales Branch could be accepted as an admirable basis. The Branch would be prepared to accept this agreement.

The Western Australian Branch had not had sufficient time to consider the matter. The Victorian Branch agreed in principle with the agreement, but desired to introduce certain modifications. The suggestion was made that a conference should be called by the Federal Committee of medical practitioners interested in friendly society lodge practice to discuss the conditions of service. The South Australian Branch was willing to adopt the New South Wales form of agreement with some modifications.

Sir George Syme thought that it would be difficult to hold a conference. The number of medical practitioners interested in lodge practice was too great. Moreover it would be necessary to devise a method for the selection of representatives. He would prefer if each Branch were asked to hold a conference of representatives of its own members engaged in lodge practice. After the views of each Branch had been ascertained they might endeavour to draft a uniform agreement either on the basis of the New South Wales agreement or a new form.

Dr. Fetherston stated that the Victorian Branch had gone into the matter and had discovered certain clauses in the New South Wales Branch agreement which it was not prepared to accept. He suggested that if the other Branches would go into the matter carefully and let them know through the Federal Committee in what way they desired to modify the New South Wales agreement, a uniform set of conditions could be drafted.

It was pointed out that as the New South Wales Common Form of Agreement had been drawn up as a compromise, the classification of the clauses were not entirely satisfactory. On the other hand the principles underlying the agreement were sound. The New South

Wales Branch would not be prepared to subscribe to a new uniform agreement unless the terms and conditions were at least as advantageous as those obtaining. Mention was made of the possible difficulty in securing the same capitation rates in all parts of Australia. Moreover it was generally agreed that the South Australian method had many advantages over the flat rate. It was ultimately agreed that the Federal Committee approved of the adoption of a model lodge agreement for all the States based on the Common Form of Agreement of New South Wales. Further consideration would be given to this matter after the Branches had had opportunity of considering it and communicating their views to the Committee.

Date and Place of Next Meeting.

It was resolved to leave it to the Chairman to determine the date and place of the next meeting of the Federal Committee.

Votes of Thanks.

Votes of thanks were carried to the Council of the New South Wales Branch for having provided accommodation for the Committee and for their hospitality and to the Chairman, Sir George Syme, for the admirable manner in which he had conducted the business of the meeting.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Medical Society Hall, Albert Street, East Melbourne, on July 7, 1926, DR. H. DOUGLAS STEPHENS, the President, in the chair.

The President introduced Drs. E. H. Molesworth and A. H. Tebbutt, of the New South Wales Branch of the British Medical Association and expressed the very great pleasure with which the Victorian Branch embraced the suggestion that two of the members of the sister Branch in New South Wales should favour them with scientific papers.

Leprosy.

DR. E. H. MOLESWORTH read a paper entitled "The Leprosy Problem" (see page 365).

DR. A. H. TEBBUTT, D.S.O., read a paper entitled "Tuberculoid Leprosy" (see page 381).

MR. H. R. DEW said that he thought that he voiced the opinion of the meeting when he congratulated Dr. Molesworth and Dr. Tebbutt on a logical and exhaustive review of a very difficult and controversial question. They had without doubt thought deeply on the matter and no one could fail to be impressed by the views put forward. With regard to the epidemiology, it certainly required much faith to attribute the disappearance of leprosy from Europe during and after the Middle Ages to the necessarily inadequate and imperfect segregation of those days. There were undoubtedly many factors in the control of the disease not yet understood and Dr. Molesworth had, he thought, clarified the whole subject. There could be no doubt that both the papers were very important contributions to the literature of this interesting subject.

The history of the disease in most foci of infection in the Pacific Islands bore out many of Dr. Molesworth's contentions. In all there had been the history of the introduction of the disease by some immigrant, usually Chinese, followed after a period of some years by an increasing number of cases until the disease became widespread throughout the population. It would seem mainly for racial and climatic reasons that the Melanesian and Polynesian peoples unlike the white races were extremely susceptible to its ravages. The rarity of cases among white traders, among missionaries or among the lowest type of whites, even when their resistance was lowered by disease or when they actually "went native," was remarkable. The tendency for the disease even among the natives to become less virulent in type after raging for many years was borne out by its history in many places.

In New Caledonia the disease had been introduced fifty-five years previously and within ten years it had spread to about half of the population, a considerable number of whites also being affected. After flourishing for many years it was found that the maculo-anæsthetic type predominated. In the Wallis Islands the only form present was an abortive type of the disease characterized by atrophy of the small muscles of the hand, anæsthesia and thickening of the ulnar nerve. The same variation in virulence of the disease had been noted in many other islands.

Important ætiological facts that must be emphasized were the extreme susceptibility of children, the necessity for close personal contact over a long period and the great length of the incubation period (five to twenty years). All observers were agreed upon the comparative rarity of infection in adults, even of conjugal infection. Mr. Dew's experience of the disease was mainly limited to Nauru, where the disease had been introduced about 1912 and had in fourteen years permeated through the island until about one-eighth of the population was affected. The degree of incidence among children was remarkable. The disease was mainly of the maculo-anæsthetic variety of a mild type, although there were a few old-standing nodular cases.

The diagnosis, as had been emphasized by Dr. Molesworth, might be extremely difficult. The earliest lesions noted in Nauru were those in children. These took the form of smooth, level, irregularly-shaped, white or fawn-coloured macules, occurring on the trunk, buttock, face or extensor surface of the limbs. In this stage there was no loss of hair, no loss of sweating and little if any anæsthesia. Sometimes the patches were definitely erythematous. A little later anæsthesia was noted, loss of appreciation of heat and cold, followed by definite depigmentation and loss of hair. Ulnar nerve thickening was noted only in a few cases. In later stages thickening of the whole area occurred and the lesion became much more like the early tubercular forms.

The diagnosis had to be made from leucoderma, tinea and psoriasis. The occurrence of anæsthesia was, he thought, all important and whenever it was seen the diagnosis of leprosy was probably correct. If the possibility of leprosy as a diagnosis were borne in mind always in any atypical skin lesion, the greater part of the difficulty vanished. The discovery of organisms in such lesions might be extremely difficult and the only way which he found satisfactory, was, as indicated by Dr. Molesworth, to excise a piece of nerve, to cut sections and to stain it. In this way a diagnosis could be made in practically all cases.

Various forms of the organism were found and this was important to emphasize. The typical homogeneous form was characteristic, but beaded and even coccoid forms of acid-fast organisms occurred and must be regarded as leprous. In these cases true lepra cells were not seen. There was a quiet cellular reaction directly under the skin with flattening of the papillæ and some depigmentation. It would seem as if there was an associated degenerative neuritis which was the cause of the anæsthesia and which spread along the nerve, ultimately leading to actual gross nerve thickening.

In regard to the variety of lesions in chronic cases he was in agreement with Dr. Molesworth and other observers who had noted all sorts of pathological changes. The tuberculoid form described by Jadassohn was an example of an interesting type which very closely resembled disseminated lupus.

There was no doubt that leprosy was often a self-healing disease and that a slow body immunity was built up, so that in many of the late macular cases even with deformities, the patient might really be non-leprous. This, as emphasized by Dr. Molesworth, was probably more likely to happen in cases occurring in non-susceptible white people. In the patient sent to him by Dr. Molesworth it seemed as if natural arrest and cure of the disease was going on and that the peculiar, chronic, low-grade granulation tissue with its endothelial giant cells was part of the curative process. Similar effects were apparently produced in patients who were improving under intravenous medication with chaulmoogra oil.

With regard to treatment, at Nauru and other leper stations in the Pacific intravenous medication was thoroughly carried out and all were agreed that it was the only satisfactory method. Both "Moogrol" and chaulmoogra oil were used with gratifying results. The vexed question of segregation was exercising the minds of all interested in the disease and many elaborate experiments on a large scale were being carried out at such places as Cullion in the Philippines, Molokai in Hawaii and Nakogai in Fiji. These combined segregation under ideal conditions (natural living conditions, few restrictions, normal activities of the patients, respect for social amenities) with rigorous intravenous medication giving the unfortunates hope of eventual cure. It must be remembered that such conditions were framed for very susceptible people in a tropical climate and that it would not be known for many years whether such methods would be efficient or whether they would be worth the enormous expense. In Nauru a similar method was being adopted with monthly inspections of the non-leprous part of the community in order to discover fresh cases.

The conditions in Australia were entirely different and Dr. Molesworth had, he thought, proved his case that in Australia segregation of such a lowly infective disease was unnecessary, ineffectual and, when the apathy with regard to such scourges as tuberculosis and venereal disease was considered, almost farcical.

Dr. Dew then moved:

> That the Commonwealth Government should take into consideration the desirability of dealing with leprosy as a national problem and that in doing so the necessity for humane methods be recognized.

The motion was seconded by Dr. Herman Lawrence who supported Dr. Molesworth in his advocacy of the discontinuance of the present system of internment, applied as it was to many lepers who could not be considered infective. It was carried.

DR. H. DOUGLAS STEPHENS, the President, said that he thought the occasion was such that the Branch might depart from its usual practice of not passing votes of thanks. Dr. Molesworth's paper had been extremely interesting and valuable and Dr. Tebbutt had demonstrated an excellent series of photomicrographs which constituted a most important contribution to the study of the pathology of leprosy. He had much pleasure in moving that the visitors be accorded a hearty vote of thanks and expressed hope that the interchange of ideas between the Branches in neighbouring States would become a regular practice.

The motion was seconded by Dr. J. Newman Morris, Vice-President, and carried with acclamation.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Mulcahy, James Edward, M.B., B.S., 1924 (Univ. Melbourne), Caulfield.

Best, John Cleveland, M.B., B.S., 1925 (Univ. Melbourne), St. Kilda Road, Melbourne.

Coffey, Frank Frederic, M.B., B.S., 1926 (Univ. Melbourne), 105, Stanhope Street, Malvern.

McCumisky, Philip Bernard, M.B., B.S., 1923 (Univ. Melbourne), Minyip.

Maling, Henry Coverley, M.B., B.S., 1925 (Univ. Melbourne), Ivanhoe.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants *locum tenentes* sought, etc., see "Advertiser," page xx.

ADELAIDE HOSPITAL: Medical Registrar.

QUEEN VICTORIA HOSPITAL, LAUNCESTON, TASMANIA: Resident Lady Superintendent.

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY: Honorary Assistant Urologist.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

SEPT. 20.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 21.—Tasmanian Branch, B.M.A.: Council.
SEPT. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 22.—Victorian Branch, B.M.A.: Council.
SEPT. 22.—Western Medical Association, New South Wales (Annual).
SEPT. 24.—Queensland Branch, B.M.A.: Council.
SEPT. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 28.—Illawarra Suburbs Medical Association, New South Wales.
SEPT. 30.—New South Wales Branch, B.M.A.: Branch: Election of two members of Federal Committee.
SEPT. 30.—South Australian Branch, B.M.A.: Branch.
OCT. 1.—Queensland Branch, B.M.A.: Branch.
OCT. 1.—New South Wales Branch, B.M.A.: Delegates of Local Associations meet Council (First Day).
OCT. 2.—New South Wales Branch, B.M.A.: Delegates of Local Associations meet Council (Second Day).
OCT. 5.—Tasmanian Branch, B.M.A.: Council.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.